AUG 14 1944

Medical Library

VOLUME 40 NUMBER 1

ARCHIVES OF

OTOLARYNGOLOGY

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

ISIDORE FRIESNER, New York
RALPH A. FENTON, Portland, Ore.
ERNEST M. SEYDELL, Wichita, Kan.
CHEVALIER JACKSON, Philadelphia
JAMES A. BABBITT, Philadelphia
J. MACKENZIE BROWN, Los Angeles
CARL H. McCASKEY, Indianapolis

JULY 1944

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1944, BY THE AMERICAN MEDICAL ASSOCIATION

CONTENTS OF PREVIOUS NUMBER

JUNE 1944. NUMBER 6

Four Decades of Nasal Allergy. Lyman Richards, M.D., Boston.

Sarcoid of the Nose. Lieutenant Commander Russell Fletcher (MC), U.S.N.R.

pH of Nasal Secretions in Situ in Atrophic Rhinitis: Its Implications. Noah D. Fabricant, M.D., Chicago.

Reconstruction of the Deformed Nasal Septum: A Critical Evaluation of the Orthodox Submucous Resection from an Anatomophysiologic Standpoint. Matthew S. Ersner, M.D., Philadelphia.

Osteomyelitis of the Frontal Bone. Lester A. Brown, M.D., Atlanta, Ga.

Recent Progress in Management of Acute Suppuration of the Middle Ear. J. R. Lindsay, M.D., Chicago.

Head Noises in Normal and in Disordered Ears: Significance, Measurement, Differentiation and Treatment. Edmund Prince Fowler, M.D., New York.

Atmospheric Pressures in the Nasal Fossa, the Maxillary Sinus and the Trachea. Lieutenant Commander Emanuel Simon (MC), U.S.N.R.

Physiology of the Nose from the Standpoint of the Plastic Surgeon. Arthur W. Proetz, M.D., St. Louis.

Role of Plastic Surgery in the Field of Otolaryngology. Samuel Fomon, M.D., New York.

Report of Isograft Transplants in Identical Twins. Alfred Schattner, M.D., New York.

Injection of the Tympanum for Chronic Conductive Deafness and Associated Tinnitus Aurium: A Preliminary Report on the Use of Ethylmorphine Hydrochloride. Barnard C. Trowbridge, M.D., Kansas City, Mo.

Perforation of Tuberculous Lymph Nodes into the Trachea and Bronchi. Oscar Auerbach, M.D., New York.

Adenoma of the Ceruminous Glands. H. J. Adler, M.D., and I. Sommer, M.D., Chicago.

Clinical Notes; New Instruments and Technics:
Use of Solution of Posterior Pituitary of Twice U. S. P. Concentration for Hemorrhage Following Tonsillectomy or Adenoidectomy. J. W. McLaurin, M.D., Baton Rouge, La.

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology.
Contributions to Plastic Surgery During 1943. Lyndon A. Peer, M.D., Newark, N. J.

Abstracts from Current Literature.

Book Reviews.

News and Comment.

Directory of Otolaryngologic Societies.

General Index.

ARCHIVES OF OTOLARYNGOLOGY

VOLUME 40 JULY 1944 NUMBER 1

COPYRIGHT, 1944, BY THE AMERICAN MEDICAL ASSOCIATION

INTRACRANIAL SUPPURATION SECONDARY TO DISEASE OF THE NASAL SEPTUM

A SURVEY OF THE LITERATURE; A REPORT OF CASES AND ANIMAL EXPERIMENTS

CAPTAIN LLOYD K. ROSENVOLD

MEDICAL CORPS, ARMY OF THE UNITED STATES

There are many causes of secondary intracranial suppurations, not a few being of primary nasal origin. Of the latter group those infections having their origin within the structure of the nasal septum will be considered in this study. During the past several decades a number of excellent reports of cases of this type have appeared in the literature, but an extensive search fails to uncover a survey in which any great number of these reports have been collected and their findings correlated. It is the purpose of this contribution to attempt to organize the data in these various reports so that definite conclusions as to incidence, causes, pathogenesis and other considerations may be drawn. The compilations of Loeb [1] were an excellent start in this direction.

As a basis for this thesis, reports of 52 fatal and 4 nonfatal cases of intracranial suppuration associated with septal infection were collected from the literature and studied.[2] It is evident that not all of these could be proved to be instances of primary septal origin and in certain instances even the actual presence of intracranial infection must be doubted. Two fatalities following submucous resection of the nasal septum were studied from the records of the White Memorial Hospital. In the case of one of these an intranasal ethmoidectomy was also done. I was privileged to see the third patient whose case I report, with Drs. T. T. Cunningham and Cyril B. Courville, who have permitted me to report it.

PRIMARY SEPTAL LESIONS

Of the primary conditions that result in intracranial extension of infection, abscess of the nasal septum and submucous resection of the septum are probably the most common. With these conditions must be considered certain intermediate associated lesions that result in intracranial suppuration. Other rare primary conditions will also be mentioned.

Abscess of the Nasal Septum.—Hirschfeld [3] studied 3,489 cases of septal abscess and found that in about 75 per cent it was secondary to trauma. A blow on the nose results in fracture of the cartilage or of the bone, usually with laceration of the septal mucosa. If a subperichondrial or a subperiosteal hematoma forms, organisms may invade the hematoma via the lacerated areas, and an abscess may then result.

Septal abscess may follow submucous resection.[4] However, in many cases this type of abscess is hardly comparable to those not associated with surgical trauma; it might better be described merely as a suppurative process between the septal flaps or in the operative wound which sometimes becomes walled off as a localized abscess. Septal abscesses of dental origin may be the result of infection of a root cyst [5] or may result from alveolar caries.[6] Septal abscess following influenza has been reported by Rudakoff [7] and others. A more rare primary cause of septal ab-

From the White Memorial Hospital and Clinic, College of Medical Evangelists, Los Angeles.

Thesis submitted to the Faculty of Otolaryngology of the University of Pennsylvania Graduate School of Medicine in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc.[Med.]) for graduate work in otolaryngology.

1. Loeb, H. W.: Fatalities Following Operations upon the Nose and Throat Not Dependent upon Anesthesia: A Study of Three Hundred and Thirty-Two Hitherto Unreported Cases, Ann. Otol., Rhin. & Laryng. **31**:273 (June) 1922; Further Studies of Fatalities Following Operations on the Nose and Throat, ibid. **32**: 1103 (Dec.) 1923.

2. Cases of otitis media as a complication following submucous resection were studied but not included in statistical considerations.

3. Hirschfeld, cited by Rudakoff.[7]

4. Hays, H.: Two Cases of Septicemia Following Submucous Resection of the Nasal Septum; One Death, One Recovery, Am. J. Surg. **23**:360 (Nov.) 1909.

5. Killian, G.: Ueber einen Fall von acuter Perichondritis und Periostitis der Nasenscheidewand dentalen Ursprungs, München. med. Wschnschr. **47**:155 (Jan. 30) 1900.

6. Helsmoortel, J., Jr.: Abcès du septum du nez d'origins dentaire, Rev. de laryng. **52**:388 (June 15) 1931.

7. Rudakoff, W. O.: Zur Kasuistik der Nasenscheidewandabszesse nach Grippe, Monatschr. f. Ohrenh. **62**:1319 (Nov.) 1928.

scesses may be any infectious process in the vicinity of the nose and its septum. Cases with this origin will not be included in this connection. A brief usable classification of the causes of septal abscesses has been given by Mangabeira Aldernaz.[8]

Intracranial complications secondary to abscesses of the septum are relatively rare. I have collected from the literature 15 fatal instances in which the septal abscess was not secondary to submucous resection. One nonfatal case has been reported by Combecher [9] and another by Unterberger.[10] In addition the former reported recovery in a case of reactive meningitis due to a septal abscess which followed submucous resection and ethmoidectomy. Watanabe [11] described a fatal septal abscess which developed two weeks after bilateral radical operation on the maxillary sinuses and radical ethmoidectomy on the left side. In spite of prompt treatment of the abscess, purulent meningitis followed, and death occurred in two weeks. In the Marburg Clinic [9] 23 cases of septal abscess (5 were apparently cases of infected hematoma) were encountered between the years 1926 and 1934. Of the patients, only 2 had meningitis of a reactive nature, and both recovered. Maxwell [12] noted no fatalities in 69 cases of hematoma and abscess of the septum observed in the Royal Infirmary of Edinburgh over a twenty year period.

Submucous Resection.—Intracranial suppuration has been recorded as a fairly direct consequence of submucous resection of the nasal septum in 27 fatal cases, including case 1 reported by me. Loeb [1] collected reports of 15 of these cases. A case with recovery is reported by Benjamins.[13] In my third case an attempt had been made to correct a septal deformity by a crushing operation.

No fatalities were recorded in 2,633 submucous resections performed during a period of twenty years at the Royal Infirmary in Edinburgh, Scotland.[12] At the White Memorial Hospital in Los Angeles at the present time about 150 submucous resections are performed each year. The two fatalities recorded in this study are the only ones on record in the hospital. They occurred in 1926. These figures seem to suggest that intracranial suppuration secondary to submucous resection of the nasal septum is quite rare.

Associated and Intermediate Conditions.—Intracranial suppuration may follow submucous resection of the nasal septum combined with other operations, such as ethmoidectomy. It is then difficult to determine from which operative wound the infection gained entrance. Loeb [1] collected 5 fatal cases of this type and I report 1. Combecher [9] reported a nonfatal case. Fabricant [14] and Powell [15] each report an instance of death following submucous resection in which the intracranial suppuration was without doubt secondary to latent posterior sinusitis that had been aggravated by the operation. The case of Risch [16] is similar. Loeb [1] reported a fatality from otitis media which was secondary to submucous resection. Houser [17] and Menger [18] each reported a nonfatal case of otitic sepsis following submucous resection. In the former's case definite thrombosis of a lateral sinus was found at the time of mastoidectomy.

REPORT OF CASES [19]

CASE 1.—*Submucous resection of the nasal septum. Early postoperative fever. Retention of nasal packing for more than forty-eight hours. Streptococcic meningitis proved present within two days after surgical intervention. Death on the third postoperative day.*

J. P., a white man aged 44, was admitted to the White Memorial Hospital on March 18, 1926. He complained of difficulty in breathing through the nose.

8. Mangabeira Aldernaz, P.: L'abcès odontogène du septum nasal, Ann. d'oto-laryng. **1**:302 (March) 1931.

9. Combecher, W.: Septumhämatome und -abszesse mit ihren Komplikationen, Ztschr. f. Laryng., Rhin., Otol. **26**:156, 1935.

10. Unterberger, S.: Grundsätzliches zur operativen Behandlung der rhinogenen Meningitis. Notwendigkeit der Wegnahme der Lamina cribrosa in bestimmten Fällen, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:193, 1934.

11. Watanabe, T.: Ein Fall von Meningitis im Anschluss an einen idiopathischen Septumabszess, Otorhino-laryng. (Abstr. Sect.), January 1940, vol. 13.

12. Maxwell, D.: Analysis of the Statistical Tables for the Years 1907 to 1926 Inclusive, Covering a Period of Twenty Years, J. Laryng. & Otol. **44**:105 (Feb.) 1929.

13. Benjamins, C. E.: Osteomyelitis der Schädelknochen bei entzündlicher Erkrankung der Nasenscheidewand, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **126**: 133 (July) 1930.

14. Fabricant, N. D.: Cavernous Sinus Thrombosis Following Submucous Resection, Arch. Otolaryng. **17**: 635 (May)) 1933.

15. Powell, W. E.: A Case of Fatal Meningitis Following Submucous Resection of the Nasal Septum, J. Laryng. & Otol. **37**:39 (Jan.) 1922.

16. Risch, O. C.: Meningitis Dependent upon Closed Ethmoiditis: Report of a Case with a Plea for Routine Autopsy Protocols, Ann. Otol., Rhin. & Laryng. **40**: 508 (June) 1931.

17. Houser, K. M.: Submucous Resection of the Nasal Septum Followed by Complications of Acute Otitis, Mastoiditis and Sinus Thrombosis, Arch. Otolaryng. **7**:631 (June) 1928.

18. Menger, L. C.: Otological Sepsis Following Submucous Resection of the Nasal Septum, Laryngoscope **42**:371 (May) 1932.

19. The first 2 cases reported were not observed by me. Permission to report them was granted by the staff member who performed the operations. Permission to report the third case was granted by Dr. T. T. Cunningham of Huntington Park, Calif.

Examination showed a nasal septum deviated to the right side, with spur formation. Aside from dental infection there were no other physical findings. Urinalysis gave normal results.

A submucous resection was done with the patient under local anesthesia. On the afternoon of the operative day his temperature as taken in the axilla rose to 101 F. The following day he rested quite comfortably, but the second postoperative day (March 20, 1926) he vomited. That evening the intranasal packing was removed. The axillary temperature reached 106.2 F. A lumbar puncture done during the night revealed turbid fluid not under increased pressure. Many pus cells were found, and culture later revealed nonhemolytic streptococci. At 8 a. m. of the third postoperative day the axillary temperature was recorded as 105.2 F.; the pulse rate was 144 and the respiratory rate 32 per minute. By noon the axillary temperature had dropped to 101.4 F. The postoperative nasal discharge was increased. Death occurred at 2:40 p. m. There was no necropsy.

Comment.—In the absence of autopsy one can only speculate as to the pathway of intracranial extension. In view of the short period of survival one can rule out retrograde thrombophlebitis. Direct extension through a traumatic rupture of the dura or extension by way of the perineural sheaths seems most likely. It is possible that retention of the postoperative nasal packing for more than forty-eight hours blocked free drainage and contributed to the spread of infection.

CASE 2.—*Submucous resection of the nasal septum and intranasal ethmoidectomy. Early, persistent severe postoperative headache. Increasing fever with chills on third day, at which time streptococci were found in cerebrospinal fluid. Death on sixth postoperative day.*

G. G., a 46 year old white man, was admitted to the White Memorial Hospital on May 3, 1926, for submucous resection of the nasal septum and intranasal ethmoidectomy. The septum was deviated to the right side, and polypi were present in the right nasal chamber. The patient had been subject to petit mal epileptic attacks for seventeen years. These had become less severe. Physical examination of the heart and lungs gave negative results. Urinalysis gave normal results.

With the operative field under cocaine anesthesia, the submucous resection was performed, and the ethmoid cells were curetted. The nasal chambers were packed with gauze.

Eight hours after the operation the patient complained of severe headache. The temperature was 100.4 F.; the pulse rate, 100 per minute. At the time the packing was removed on the following morning, the temperature was only 99.6 F. He continued to complain of severe headache. The temperature reached higher peaks each day, and on the third postoperative day, when he had some chills, it reached 105.8 F. Lumbar puncture revealed many pus cells in the cerebrospinal fluid, and culture showed nonhemolytic streptococci. The next day the axillary temperature was 105.8 F. The cerebrospinal fluid was under greatly increased pressure, as evidenced by its shooting over the top of the manometer during lumbar puncture. It still contained many pus cells and nonhemolytic streptococci.

The headaches continued, the patient became delirious at times and he also had some "epileptic seizures." Two hours before death the axillary temperature was 105.8 F., the pulse rate 168 and the respiratory rate 70. Autopsy was not permitted.

Comment.—The infection could have had an intraceptal origin or an origin in the ethmoid sinus wound. The pathway of extension was possibly not direct as through a dural laceration. Loeb[1] expressed the belief that when the onset of meningitis occurs after the second day the infection is usually not associated with a basal fracture or a dural laceration but spreads to the meninges in another way.

CASE 3.—*Crushing of nasal septum with forceps in attempt to straighten septum. Subsequently headaches and mental disturbances, followed by reactive meningitis within three weeks. External frontal sinus opened for purulent sinusitis. Patient improved except for low fever. Two months later submucous resection of septum, followed by high fever and evidence of meningitis, with death in five days.*

S. M., a 25 year old man, had injured his nose at the age of 13, and since that age his right nostril had been obstructed. On Feb. 13, 1937 a general practitioner tried to straighten the nasal septum by crushing it with forceps. There is also some question of partial turbinectomy having been done. Following the "operation," the patient complained of frontal and bitemporal headaches. On March 2 the headaches became more severe, and the neck began to be stiff. Mental disturbances developed, such as failure to recognize his wife.

Because of the obvious seriousness of the patient's condition, he was referred to Dr. T. T. Cunningham, who in turn obtained neurologic consultation with Dr. Cyril B. Courville. I was also privileged to see the patient as an assistant to Dr. Courville. On March 5, there was definite evidence of meningeal irritation, and the spinal fluid, which was under increased pressure, contained 1,200 cells per cubic millimeter, 40 per cent of which were polymorphonuclear. Because of roentgen evidence of right frontal, ethmoid and maxillary sinusitis, the right frontal sinus was explored through an external approach by Dr. Cunningham on March 6. The sinus contained hemorrhagic mucopurulent material which was under obvious pressure. Exposure of the dura revealed no extradural infection. The patient improved greatly after this operation with the exception that a temperature of 99 F. to 100 F. persisted. On March 13 the spinal fluid count was 30 mononuclear cells per cubic millimeter. About May 13 severe frontal and vertical headache developed.

On May 17 submucous resection was performed. Following the operation a septic type of fever was noted, and on May 20 the temperature reached 104 F. There was definite evidence of meningeal irritation but no localizing signs. Five days after submucous resection the patient died. Permission for autopsy was not granted.

Comment.—In retrospect it becomes clear that in this patient latent meningitis developed following the original crushing operation on the septum, which also probably fractured the cribriform plate. Since there were no localizing signs correct diagnosis was difficult. The operation on the frontal sinus and the subsequent resection of the septum seemed indicated in view of the clinical situation at the time. Following the last operation, the latent meningitis about the cribriform plate flared up into the fatal issue.

PATHWAYS OF INFECTION

With infection of the nasal septum there may be invasions of the intracranial space by adjacent or regional pathways and by distant or indirect pathways. The former are direct extensions (1) through traumatic dehiscences, (2) through natural bony passages, (3) through bony de-

hiscences consequent to osteomyelitis, (4) through veins by retrograde thrombophlebitis and (5) via the perineural sheaths of the olfactory nerves. The distant or indirect pathways are represented by (1) the general circulatory system (septic pathway) and (2) neighboring organs with intermediate infection as a result of septal disease, e. g., otitis following submucous resection.

Direct Extension Through Traumatic Dehiscences.—This mode of invasion, as far as reports in the literature are concerned, seems to be more rare than expected. Loeb [1] secured the report of 1 case of meningitis following submucous resection in which autopsy revealed a puncture of the cribriform plate. In another case, without autopsy, the patient died of meningitis two days after the operation, and the reporter was quite sure that a fracture was responsible. Another of Loeb's collected cases was one in which the cribriform plate was fractured and cerebrospinal fluid escaped during the submucous resection and intranasal frontal and ethmoid operations. Loeb expressed the belief that if meningitis develops during the first day after operation the extension of infection is probably on the basis of trauma.

Miodowski [20] reported a fatal case of meningitis following submucous resection in which at autopsy a splinter of bone in the region of the crista galli was given consideration as being a contributory factor in the pathogenesis. However, no dural tear was found, and the mode of extension assigned in his case was infection of the olfactory perineural spaces. Ertl [21] considered the possibility of fracture of the cribriform plate during septal trauma allowing passage of infection into the cranium. However, there was no postmortem evidence to support this consideration. Infection of a perineural sheath was also considered. Cordeiro and Braz Gravina [22] in their report of a fatal case of meningitis following a traumatic septal abscess suggested the possibility of a fracture of the cribriform plate as a factor in such cases. This, however, seems rather unlikely in their case, in which no autopsy was performed.

Whatever the role of traumatic dehiscences may be in connection with meningitis of septal origin, extension by way of them can no doubt result whether the dura is torn or not; in most cases it no doubt is torn. Some are so reported. If the dura mater is not lacerated but the cribriform plate is fractured, infection of the extradural space may eventually extend to the intradural structures.

Extension Through Natural Bony Passages.—Such passages as the natural openings in the cribriform plate can be considered as pathways for intracranial extension. It is quite unlikely, however, that this extension often occurs by direct continuity and ulceration of tissue. It is more likely associated with retrograde thrombophlebitis of the perforating vessels or infection of the sheath of an olfactory nerve. The openings in the cribriform plate in their natural state are filled with soft tissues and are not empty holes as in a dry skull through which one might imagine pus or other fluids passing easily.

Extension through congenital bony defects in the cranial wall is no doubt rare in connection with septal complications. In fact, no such case has been reported. Froboese [23] reported a congenital dehiscence of the cribriform plate. The meningitis which developed by way of this opening was not of septal origin however.

Carter [24] cited 2 fatal cases of meningitis following traumatic septal abscesses which he claimed extended via the cribriform plate. Dabney [25] also mentioned a fatal case of cerebrospinal meningitis occurring one week after submucous resection in which the infection was thought to have traveled through the cribriform plate. Unterberger [10] recorded in detail a case previously reported statistically by Kindler.[26] The patient was a child with a post-traumatic septal abscess. When signs of meningitis developed, a modified submucous resection was done and the cribriform plate exposed. Both ethmoid labyrinths were also exenterated for reasons not stated. Autopsy twenty-four hours later revealed an extradural abscess over the lamina cribrosa, and the septal abscess was found to communicate freely with the epidural abscess through a hole in the lamina cribrosa. It is

20. Miodowski, F.: Komplikationen nach submukoser Septumresection, histologische Untersuchungen über ihre Genese, Verhandl. d. internat. Laryng.-Rhin.-Kong. **5**:179, 1912.

21. Ertl, E.: Hirnabszess infolge von Pfählungsverletzung der Nase, Monatschr. f. Ohrenh. **69**:429 (April) 1935.

22. Cordeiro, H., and Braz Gravina: Sobre un caso de abcesso do septo nasal post-traumatico complicado de meningite, Rev. oto-laring. de São Paulo **1**:376 (Sept.-Oct.) 1933.

23. Froboese, C.: Missbildung der Lamina cribrosa des Os ethmoidale als Ursache der eitrigen Meningitis, Berl. klin. Wchnschr. **54**:1219 (Dec. 17) 1917.

24. Carter, W. W.: Abscesses of the Nasal Septum: Their Etiology and Treatment with Reference to Resulting Deformities, M. J. & Rec. **119**:11 (Feb. 6) 1924.

25. Dabney, V.: Deaths Attributable to Intranasal Operations and Other Instrumentation, Surg., Gynec. & Obst. **22**:324 (March) 1916.

26. Kindler, W.: Die rhinogene Meningitis: Nach 9 jährigen Erfahrungen an der Grazer Klinik (1922-1930), Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **29**:309 (Jan.) 1932.

not stated whether this hole was due to disease or was one of the natural olfactory openings. In one area an olfactory nerve traversed the extradural abscess. There was also beginning osteomyelitis of the frontal bone incident to the extradural abscess. The osteomyelitis had eroded the orbital roof and started a subperiosteal orbital abscess. Since the osteomyelitic focus was in relation to the canal for the anterior ethmoidal nerve, this preformed pathway may, according to the author, in some way have been connected with the spread of infection to the orbit. If the hole in the cribriform plate noted by him was one of the natural openings, this instance can be considered as one of true extension via a natural bony passage.[27] If the opening described was a result of the infection, one must consider either ulcerating osteitis or focal erosion of the bone.

Extension by Means of Intervening Osteomyelitis.—Benjamins [18] reported 3 cases of cranial osteomyelitis following septal infection. The first case was that of a child in whom a septal abscess developed following a common cold. On incision and drainage it was found that a part of the bony septum was absent, presumably as a result of erosion. A swelling also developed in the forehead, and pus was evacuated over the medial part of the right eyebrow by incision. At autopsy, erosion of the outer table of the skull over the medial aspect of the right brow was noted. The dura was covered with pus, and a large subdural abscess was present on the left side. The extradural and subdural abscesses communicated through ulcerations in the dura. The canals of Breschet in the cranial vault were filled with pus. All of the paranasal sinuses were normal. There were also multiple abscesses of the lung.

Benjamins' second case was one of osteomyelitis of the frontal bone and extradural abscess following submucous resection of the nasal septum. The operation was complicated by acute angina and a high fever. The osteomyelitis was cured by radical removal of the bone in the right frontal region. The right frontal sinus was opened and found to be normal, proving that the bone infection did not originate in the sinus but originated from the septum operated on. His third case was one of septal abscess following influenza. There was swelling of the root of the nose, the right side of the forehead and the eyelids. No free pus was found in the nasal chambers, and the air sinuses appeared normal on examination. Evacuation of the septal pus and application of a pressure bandage over the swollen areas resulted in recovery. (I doubt that in this case there was fully developed osteomyelitis.) In discussing the pathogenesis, Benjamins ruled out metastasis and indirect infection by way of the superior sagittal sinus. He said that it is more likely that the transmission was by local blood vessels as in thrombophlebitis. Dreschel [28] is credited with having demonstrated that in the newborn infant there is a direct blood vessel connection between the upper part of the nasal septum and the frontal bone. It is not known whether the vascular connection is as well developed in adults. Two reasons are suggested [18] to explain the rarity of cranial osteomyelitis originating from the nasal septum. The first is that the infection must be present high in the septum for osteomyelitis to occur. The second is that it is possible that the vascular connection described by Dreschel is present only in rare or few instances. When one considers that most septal abscesses (and also hematomas) are located in the cartilaginous or lower and anterior part of the septum, the first reason attains a certain degree of plausibility. It would seem fitting at this juncture to postulate a third reason for the rarity of cranial osteomyelitis following septal infection. That reason may be that the perpendicular plate of the ethmoid bone, which constitutes the upper part of the bony nasal septum, is generally very thin and compact, containing little marrow except near its articulations and, being made up largely of cortical bone, containing few vascular channels in which thrombophlebitis can begin and be transmitted.

Unterberger's case [10] in which osteomyelitis was a possible factor in the intracranial extension of a septal abscess has already been described. Jacobsgaard [29] in discussing the general problem of the genesis of rhinogenic meningitis mentioned "perforating osteomyelitis." He

27. A critical study of case reports in which infection is said to have extended directly from the septum to the intracranial space through the holes in the lamina cribrosa reveals that the allegation is not always supported by autopsy or histologic evidence. It would seem, therefore, that extension by this route is actually quite rare. Turner and Reynolds [41] gave no consideration to direct extension through the natural openings of the cribriform plate, considering only three main modes of extension from the nose to the intradural structures, namely, (1) direct extension through bone, (2) infective thrombosis of the venous system and (3) extension by way of the olfactory perineural sheaths. They naturally also recognized direct extension as a result of trauma.

28. Dreschel, cited by Benjamins.[18]

29. Jacobsgaard, J.: Pathologisch-anatomische Beiträge zur Frage nach den Fortleitungswegen der rhinogenen Meningitis, Ztschr. f. Laryng., Rhin., Otol. **26**: 387, 1936.

does not seem to favor a thrombophlebitic spread of osteomyelitis. Perhaps by "perforating osteomyelitis" he means ulcerating osteitis or focal erosion.

Rabbit 2 in my experimental series would seem to illustrate osteomyelitis as a means of extension of infection. However, in this animal as in clinical cases of osteomyelitis the ultimate or real means of extension is often a thrombophlebitic process usually of a retrograde nature.

Extension by Retrograde Thrombophlebitis.—There can be little doubt that this means of spread of infection intracranially from the various structures of the head is being given more and more prominence by various investigators.[30] Morwitz [31] mentioned meningitis secondary to septal abscess as being transmitted via the ethmoid blood vessels. In Fabricant's[14] case of thrombosis of the cavernous sinus following submucous resection of the nasal septum the suppuration was thought to have extended from infected sphenoid and ethmoid sinuses to the cavernous sinus by way of the ethmoid veins. Loeb,[1] in analyzing 15 cases, expressed the belief that meningitis following submucous resection may in part be of "hematogenous" transmission. Whether the term he used can be regarded as synonymous with "thrombophlebitic" is not clear, but general sepsis is discussed by him as a separate group. In the case of cavernous sinus thrombosis following a septal abscess reported by Goldman [32] the infection may have reached the sinus by way of thrombosis of the intervening veins. In the case of cavernous sinus thrombosis following a traumatic septal abscess reported by Clay [33] the spread was no doubt by retrograde thrombophlebitis involving the ethmoid and orbital veins. There was no autopsy. As mentioned earlier, Benjamins [18] seemed to admit that thrombophlebitis is a likely means of spread of infection from the septum. Kramer and Som [34] have attached relatively minor importance to spread by venous channels as it pertains to the sphenoid and ethmoid sinsuses. However, they did express the belief that it is a prominent factor in acute frontal sinusitis associated with osteomyelitis.

The vessels most likely to be involved in thrombophlebitis would be the veins that pass with the olfactory nerves through the cribriform plate and in rare instances vessels within the septal marrow. The ethmoid veins might also be involved as pathways of extension from the septum. Thrombophlebitis of the minute perforating veins can be detected best in microscopic sections. Consequently it is difficult to ascertain pathways of infection in fatal cases that are not subjected to necropsy. In the animal series herein described, rabbits 2, 3 and 7 demonstrate a thrombophlebitic type of extension of infection from the nasal septum to the intradural structures.

Extension Along the Perineural Spaces of the Olfactory Nerves and the So-Called Perineural Lymphatics.—Probably no term used to designate a pathway for the intracranial extension of nasal infection has been more abused than "the lymphatic route." No doubt the lymphatic pathways in and about the head do play a part in the spread of infection intracranialward but not to the extent that writers both past and present would have one believe. Courville and I [35] reviewed some of the background of this problem and pointed out that infections which leave no gross trace may have "traveled by way of the perineural sheaths or the communicating venous channels."

The perineural spaces are not true lymph spaces. They are prolongations of the subarachnoid space,[36] and as such their fluid content is cerebrospinal fluid and not lymph. Jacobsgaard,[29] as a consequence of his histopathologic studies and survey of the literature, stated that the olfactory nerve sheaths and the fine lymphatics of the nasal mucosa travel through the cribriform openings side by side. He expressed the belief that the olfactory "lymphatics" are important in pathogenesis whereas the ordinary lymphatics are not important. In his histologic study of 6 cases of rhinogenic meningitis without surgical intervention he found that in 4 the

30. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **16:** 203 (Aug.) 1932.

31. Morwitz, S. M.: Acute Abscess of the Nasal Septum: Case Reports, Illinois M. J. **43:**463 (June) 1923.

32. Goldman, J. L.: Cavernous Sinus Thrombosis Following Intranasal Infections, J. Mt. Sinai Hosp. **2:**121 (Sept.-Oct.) 1935.

33. Clay, J. V. F.: Abscess of the Nasal Septum Followed by Orbital and Cavernous Sinus Infection, J. Ophth., Otol. & Laryng. **26:**254 (July) 1922.

34. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32:**744 (Oct.) 1940.

35. Courville, C. B., and Rosenvold, L. K.: Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses, Arch. Otolaryng. **27:**692 (June) 1938.

36. D. Novelli (Sulle communicazioni fra spazi subaracnoidei e mucosa nasale, Boll. d. mal. d. orrechio, d. gola, d. naso **54:**131 [April] 1936), in studying the subarachnoid prolongations along the olfactory nerve filaments, injected various dyes subdurally in animals and later found them deposited along the olfactory nerve sheaths. His injections were made in the lumbar as well as in the cisternal regions and no doubt were actually made into the subarachnoid spaces.

pathway of extension was via the olfactory nerve sheaths. In the remaining 2 cases the infection extended by way of osteomyelitic processes.

Kramer and Som [34] traced a perivascular lymphatic infiltration from the lining of the sphenoid sinus through the bone to an extradural abscess. They apparently recorded no further extension of the lymph structures. Indeed, I have yet to see any report in which lymph vessels are clearly and unquestionably demonstrated to pass through the dura into the subarachnoid space.[37] It is further stated [34] that:

Can one not legitimately suggest that perhaps the alleged communication as well as the function is also more apparent than real?

Kramer and Som[34] studied histologically 1 case in which it was proved that the infection reached the subarachnoid space by way of the perineural sheaths. Their statement that "except in rare instances infection does not spread through these perineural sheaths to the subarachnoid spaces" is not fully supported by the available literature.[38] This is especially true when one considers how infrequently serial microscopic studies are made and that it is only by microscopic means that this pathway can be properly established. Brunner [39] studied microscopic serial section from a fatal case of rhinogenic meningitis and found a purulent type of infiltration in the olfactory mucous membrane of the septum and the middle turbinate on the affected side. He definitely stated that the infection traveled upward in the olfactory nerve sheaths of these areas. Miodowski [20] made similar studies of 2 fatal cases of meningitis following submucous resection of the nasal septum. Pus was found in the perineural spaces of the olfactory nerves, and as proof against the pus being of retrograde origin from above, he pointed out that the pus was found only in connection with the olfactory filaments of one side in each case..[40]

Reference [29] has already been made to 4 other cases of demonstrated extension along the perineural spaces. Turner and Reynolds [41] also described 4 cases of rhinogenic meningitis in which the infection was proved to have passed to the meninges within the perineural sheaths. In addition they referred to 2 similar cases reported by von Eicken in which the perineural pathway was traced with the aid of the microscope. Hofmann [42] studied a fatal case of meningitis and intracranial arterial hemorrhage following a septal abscess. Grossly, red streaks were visible along the olfactory filaments. Microscopically, punctate hemorrhages were noted on the septum, and a purulent infiltration was present to some extent in the perineural spaces. The latter was also true to a lesser extent on the lateral nasal wall where olfactory nerve filaments were located. Hofmann definitely expressed a belief that the perineural spaces transmitted the infection to the meninges.[43]

37. A. Key and G. Retzius (Studien in der Anatomie des Nervensystems und des Bindegewebes, Stockholm, Samson & Wallin, 1875-1876, vol. 1, p. 217) were among the earliest investigators to postulate a lymphatic connection between the nose and the subarachnoid space. Schwalbe and Andre (cited by Jacobsgaard [29]) and Beck (cited by Berendes [45]) also favored the lymphatic connection. Indeed, H. Zwillinger (Zur Frage der auf dem Wege der Wege der Lymphbahren enstehenden intrakraniellen Komplikationen nasalen Ursprunges, Monatschr. f. Ohrenh. **46**:675, 1912) injected dyes into the subarachnoid spaces of fresh cadavers and believed that he found a direct connection between the olfactory perineural spaces and the superficial lymphatic vessels of the nasal mucosa in the olfactory region. He also described a lymphatic connection between the frontal sinuses and the subarachnoid space. Novelli in his parallel studies on live animals did not trace the dye farther than the perineural spaces. Turner and Reynolds [41] stated that "direct continuity between the lymphatic vessels of the peripheral parts and the intradural structures—the leptomeninges and brain—does not exist." They do, however, recognize peripheral lymphatic vessels as taking origin in the internal layer of the dura mater. I believe that though the conclusions of Zwillinger and others cannot be refuted by mere words, the clinical significance of their deductions has been greatly overestimated.

The perineural lymph channels surrounding the filaments of the olfactory nerve communicate with the subarachnoid spaces endocranially and with the superficial network of lymphatics of the olfactory mucosa intranasally. This free lymphatic communication should appear to be an open avenue for the spread of infection to the meninges. However, experience has proved that function is more apparent than real.

38. Disagreement with certain authors on this point is not intended to detract from or discredit the fine work done by those authors.

39. Brunner, H.: Zur Pathologie und Klinik der rhinogenen Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **157**:92, 1937.

40. It is of course possible in meningitis for pus cells to sink into the olfactory nerve sheaths, but in instances of this the pus cells were never found below the level of the cribriform plate.[20] This deduction is based on control cases of meningitis of other than rhinogenic origin. A similar finding has also been recorded by other authors (Turner and Reynolds [41]; Kramer and Som [34]).

41. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Disease, Edinburgh, Oliver & Boyd, Ltd., 1931.

42. Hofmann, L.: Ein Fall von Nasenscheidewandabszess mit konsekutiver Meningitis und Ruptur einer Hirnaretrie, Monatschr. f. Ohrenh. **66**:1409 (Dec.) 1932.

43. I wish to suggest that the grossly visible red streaks along the olfactory filaments and the microscopic hemorrhages possibly indicated an associated vascular type of extension, such as a retrograde thrombophlebitis.

In addition to 3 cases [44] of primary septal infection traced intracranially along the perineural sheaths in microscopic studies, there is a case with autopsy reported by Ertl [21] in which the olfactory nerve sheath was considered as a pathway of extension. In a case with autopsy studied by Berendes [45] the infection was thought to have extended intracranially along the "lymphatics" as in some of Loeb's [1] collected fatal cases. The olfactory perineural lymph spaces were looked on as the pathway of extension for the fatal meningitis which followed the septal abscess described by Watanabe.[11] There was no autopsy.

In none of the rabbits of my short experimental series could evidence be found of lymphatic or even perineural extension of infection intracranialward. In rabbit 7, however, there was evidence of a thrombophlebitic process in the blood vessels situated parallel and adjacent to the olfactory nerve sheaths.

To summarize, it can be stated that infection by way of the olfactory perineural spaces has been shown to occur as reported by several authors. In at least 15 cases with autopsies has this pathway been traced by microscopic evidence; in 3 the nasal septum was the primary site of infection. The burden of proof still rests with those who insist that intradural extension along lymph channels of the ordinary kind is possible. Turner [46] lent little support to the lymphatic theory of extension. He cited the case of Hayen in which ferric chloride applied as a nasal tampon was demonstrated after death in the olfactory nerve substance. He concluded that "it was evident that the perchloride or iron had reached the brain along the nerve branches passing through the cribriform plate." This description can be interpreted only as referring to the olfactory nerves and their perineural sheaths and is in full consonance with Turner's more recent statement [37] that direct continuity between the peripheral lymphatics and the intradural structures does not exist. The small perforating blood vessels accompanying the olfactory nerve sheaths through the cribriform plate should not be overlooked as possible transmitters of infection even though apparent disease is noted along the perineural sheaths.

Indirect Intracranial Extension by Way of Septicopyemia.—In considering sepsis in relation to intracranial extension of infection from the nasal septum, one may recognize three possibilities: First, septicopyemia may be a means of, or a pathway for, the transmission of the infection; second, it may be merely a manifestation of a more direct invasion by thrombophlebitis of the intracranial venous channels, such as the cavernous sinus; third, septicopyemia may be primarily of septal origin with infection extending intracranially independently of the septic pathway. A clinical diagnosis of the last type would seem to be difficult to attain and is only of theoretic interest and possibility.

Evans [47] reported a cortical cerebral abscess following a submucous resection of the nasal septum. He expressed the belief that the abscess was the result of a pyemic type of spread from the septum. He also reported an abscess of the temporal lobe following frontal sinusitis as resulting from pyemia. In the fatality following submucous resection that was reported by Errecart [48] clinical signs of meningitis developed as well as a rash involving mostly the extremities. The operative day passed without incident, but on the following day high fever and other fatal signs appeared, and the patient died on that same day. Sore throat also developed, and the author was not sure what role that complication played. In the absence of an autopsy one can surmise that the meningitis was probably the result of severe sepsis, of which there were also clinical signs.

Kelemen [49] reported a case of submucous resection without local complications. The second day after surgical intervention the patient noted a severe pain of the leg on arising from his bed. Swelling of the calf of his leg was detected. Two days later there was more pain, and at that same time there were also clinical and roentgenologic signs of a pulmonary infarct. Recovery ensued. There can be little doubt that the embolic thrombi probably originated in connection with the nasal wound. The emboli were probably not infected, but this case is cited to illustrate that it might be possible for infected emboli to result following submucous resection.

General sepsis following submucous resection is not unknown. Loeb [1] reported 3 fatal cases. One of the patients died as late as nine days after the operation. Septic or pyemic manifestations without death have been reported following sub-

44. Miodowski,[20] Hofmann.[42]

45. Berendes, J.: Stirnhirnabscess als Folge eines Septumabszesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:148, 1934.

46. Turner, A. L.: The Spread of Bacterial Infections from the Nasal and Nasopharyngeal Cavities by Way of Lymphatic Channels, Edinburgh M. J. **7**:409 (Nov.) 1911.

47. Evans, W.: The Pathology and Etiology of Brain Abscess, Lancet **1**:1231 (June 6) 1931; 1289 (June 13) 1931.

48. Errecart, P. L.: Operación de Killian por deformación del septum nasal: Exitus, Rev. Asoc. méd. argent. **50**:974 (June) 1936.

49. Kelemen, G.: Thrombose und embolischer Lungeninfarkt nach submuköser Septumresektion, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:139 (July 3) 1930.

mucous resection in 3 cases [50] and following simple septal abscess in 1 case.[51] No doubt mild clinical manifestations of sepsis are frequently encountered as a complication of operations and abscesses on the nasal septum.

Fabricant [14] and Hays [4] each reported a fatal case of cavernous sinus thrombosis and meningitis following submucous resection. Autopsy studies made in 1 case [14] revealed a purulent state of the ethmoid and sphenoid sinuses. It was thought that the ethmoid veins may have been the avenues of transmission for the infection. Whether the nasal septum or an air sinus was the primary site of infection could not be proved. The septicemia described in each case and proved present by blood culture in Fabricant's case was no doubt secondary to the thrombosis of the cavernous sinus. The extension of infection from the septum to the intracranial structures was doubtless a local process and did not occur by way of septicopyemia.

Thus in review there seems to be recorded only 1 case (that of Evans [47]) of intracranial infection of septal origin as to which one can be reasonably certain that the pathway of extension was by septicopyemia. I am unaware of any detailed microscopic studies having been done in this lone case to disprove that any other pathway of local extension was possible. It is to be regretted that the case of Errecart [48] was not subjected to autopsy in an effort to corroborate the clinical evidence of meningitis of septic origin which followed submucous resection.

Indirect Adjacent Pathways of Intracranial Extension.—With regard to the case of cavernous sinus thrombosis reported by Clay [33] there is no doubt that the spread was by way of veins in the orbit. A further illustration of how infection from the septum may reach the orbit is furnished by Mahoney's [52] report of a retrobulbar abscess as a consequence of submucous resection and simple infraction of the middle turbinate. When the deep orbital pus was evacuated, no communication with the nose was found. It is likely that thrombophlebitis was the means of transmission.

Risch [16] recorded a case in which submucous resection was soon followed by acute tonsillitis, otitis media and mastoiditis in this order. Despite myringotomy and finally mastoidectomy, the patient died of meningitis, which was thought to be due to the mastoid infection. However, at autopsy the leptomeningeal pus was definitely traced to the cribriform area of the right side. The right ethmoid sinuses were filled with pus, and Risch expressed the belief that they were the source of the meningitis. Powell's [15] patient died twelve hours after submucous resection from acute fulminating meningitis. Autopsy revealed severe ethmosphenoiditis with some evidence of osseous damage from the suppuration. There was no operative trauma at the base of the skull. In Fabricant's [14] case likewise latent ethmosphenoiditis was found only at autopsy. These cases seem to indicate that deep-seated infections of the air sinuses, possibly with beginning extension, are stimulated to further activity and extension by submucous resection. The factors involved are no doubt local obstruction of drainage and a general lowering of resistance by the operation.

Brühl [53] reported a septal resection in a young woman following which there occurred a severe flare-up of acute frontal sinusitis with death ensuing three days after onset of the acute symptoms. At autopsy the right frontal sinus was filled with pus. There was thrombosis of the superior sagittal sinus with suppuration and purulent meningitis. The reporter concluded that the operation on the nasal septum with postoperative packing and blockage of drainage from the frontal sinus had permitted a flare-up of the sinusitis.[54] The infection then spread from diploic veins to the dural veins and thence to the superior sagittal sinus and finally to the leptomeninges. Halle,[55] however, took issue with

50. Carando, V.; Galante, E., and Robbio Campos, J.: Complicaciones de la resección submucosa del tabique, Semana méd. **1**:225 (Jan. 21) 1937. Hays.[4] Miodowski.[20]

51. Culbert, W. L.: Report of a Case of Bilateral Abscess of the Septum with Well Marked Symptoms of Septicemia and Report of a Case of Epiglottic Abscess, with Secondary Involvement of the Cervical Glands, Ann. Otol., Rhin. & Laryng. **12**:293, 1903.

52. Mahoney, P. L.: Retrobulbar Abscess Complicating Submucous Resection, Laryngoscope **42**:199 (March) 1932.

53. Brühl, G.: Kasuistischer Beitrag zu den rhinogenen endokraniellen Komplikationen, Monatschr. f. Ohrenh. **64**:1061, 1930.

54. An interesting example of long-standing blockage of the duct of the frontal sinus is afforded by the case reported by H. M. Goodyear (Osteoma of the Nasal Septum with a Large Epidural Abscess; Operation; Recovery, Arch. Otolaryng. **14**:23 [July] 1931) in which an osteoma of the upper part of the septum was removed through a lateral rhinotomy. Pus was found in the frontal sinus, and further exploration revealed absence of part of the posterior wall of the sinus as well as of the anterior third of the cribriform plate. Granulations were present on the dura. The extradural abscess was primarily secondary to the frontal sinusitis and was related to the septal tumor only in that the latter no doubt contributed to obstruction of drainage from the frontal sinus, thus favoring continuance of the sinusitis. This same association is seen in cases of marked deflection of the septum with consequent obstruction to sinus drainage.

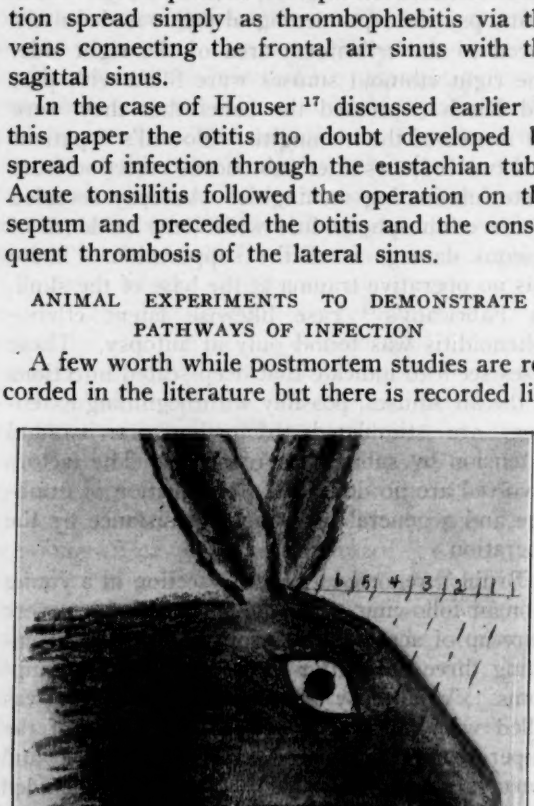
55. Halle, M.: Zu den rhinogenen endokraniellen Komplikationen, Monatschr. f. Ohrenh. **65**:425 (April) 1931.

these conclusions and assigned little or no direct connection between the operation on the septum and the spread of the infection from the frontal sinus. He expressed the opinion that the infection spread simply as thrombophlebitis via the veins connecting the frontal air sinus with the sagittal sinus.

In the case of Houser [17] discussed earlier in this paper the otitis no doubt developed by spread of infection through the eustachian tube. Acute tonsillitis followed the operation on the septum and preceded the otitis and the consequent thrombosis of the lateral sinus.

ANIMAL EXPERIMENTS TO DEMONSTRATE PATHWAYS OF INFECTION

A few worth while postmortem studies are recorded in the literature but there is recorded little if any experimental work with animals in which attempts were made to ascertain the pathways of extension of septal infection. In an attempt to clarify the genesis of the fatal complications, the nasal septums of 7 rabbits were inoculated with virulent organisms. Pneumococci were used in 6 rabbits and Staphylococcus aureus in 1. Five of the 6 rabbits inoculated with pneumococci died; in 4 the meninges yielded the identical inoculated organisms in the correct type on culture, and in 1 of these 4 the heart blood also contained the organisms. The skulls of the rabbits that died were sectioned (figure) and studied histologically. The protocols follow:



Location of sections taken from skulls of experimental rabbits.

RABBIT 1.—With the animal under ether anesthesia, an incision was made in the left side of the columella and the mucoperichondrium and the mucoperiosteum were elevated from the septum as in submucous resection on the human being, and 0.25 cc. of virulent type III pneumococci suspended in isotonic solution of sodium chloride was deposited in the septal cavity high up and posteriorly. The anterior incision was closed by packing cotton in the naris. The animal did not die and showed no ill effects from the procedure.

RABBIT 2.—With the animal under ether anesthesia, 0.1 cc. of a saline suspension of type 19 pneumococci was injected into the septal mucosa of the left side high and posteriorly by means of a long needle. By proper measuring and probing I assured myself that the injection was not made intracranially. The same injection technic was used in the remaining rabbits of the series. (The pneumococci were isolated by culture from a case of pneumonia with meningitis that later proved fatal.)

The animal died in less than forty-eight hours. Autopsy under aseptic technic revealed a normal gross appearance of the meninges. Culture of the leptomeninges produced a growth of type 19 pneumococci. The heart blood was sterile.

After the head had been fixed in a solution of formaldehyde, decalcified and embedded in paraffin, sections were made of the nasal fossae and anterior part of the brain and brain case. The accompanying figure shows the blocks taken for sectioning. The microscopic sections were stained with hematoxylin and eosin and by the Gram-Weigert method. The procedure and technic used were essentially the same in all of the autopsies on these animals.

In section 2 of this rabbit engorgement of the septal vessels on the injected side was noted. Some inflammatory exudate was present in that nasal chamber. Pneumococci were visible in the blood vessels of the septal and the turbinal mucosa as well as in the nasal exudate. Section 3 showed essentially the same pathologic features as section 2.

Section 4 revealed engorged vessels in the nasal roof. The marrow, meninges and brain were hyperemic. The marrow and the meninges showed large hemorrhages. Diplococci were visible in the hyperemic vessels and hemorrhages. Some of the vessels of the brain contained organisms. The general appearance of the bone and marrow suggested the beginning of osteomyelitis. One section showed the beginning of a bony canal through which the organisms could gain entrance from the nasal mucosa. No inflammatory reaction or organisms could be demonstrated about the olfactory nerve filaments.

Comment.—This animal demonstrated the transmission of a septal infection to the meninges by a process of retrograde thrombophlebitis. Septicemia must be considered, but the sterile heart blood helps rule this out. The depositing of the suspension of organisms into a large septal blood vessel at the time of the injection is possible but not probable.

RABBIT 3.—The same organism and technic of injection were used as in rabbit 2. The animal died in less than six days. At autopsy the pneumococci were isolated from the meninges. The heart blood was sterile. Grossly, the leptomeninges of the anterior fossa were hyperemic. Some gross suppuration was noted.

Sections of the several levels revealed vegetation on the septal mucosa on the side of injection. Pneumococci were demonstrated in the vegetation by the Gram-Weigert method. There was engorgement of the vessels of the same side. Inside the skull there were areas of hemorrhage and some exudation. The brain tissue seemed edematous. No organisms could be demonstrated intracranially.

Comment.—This animal showed striking vascular dilatation and hemorrhage in the vicinity of the infectious lesions. Unfortunately, though the meningeal culture was positive, no organisms could be demonstrated in the sections of meninges that were stained. The hemorrhagic features point to a thrombophlebitic process. No perineural changes were recognized.

RABBIT 4.—Two-tenths cubic centimeter of a peptone broth suspension of type 18 pneumococci was injected into the nasal septum. The culture had been obtained from a human case of postoperative pulmonary atelectasis. The animal died in less than thirteen days. Autopsy revealed slightly opaque meninges. Cultures of meninges and heart blood both gave growth of type 18 pneumococci in pure culture.

Coronal sections of the head revealed only a small "denuded" area of mucosa near the roof of one nasal chamber. Some exudation was seen in the nasal chambers. The forebrain showed some vacuolation. No organisms could be demonstrated in any of the sections.

Comment.—In view of the few local findings and the culture of the injected organisms from both the meninges and the heart blood, one must consider a blood-borne (septicemic) transmission of the infection. The long period of survival might, however, be an argument against a septicemic or pyemic spread.

RABBIT 5.—Two-tenths cubic centimeter of a saline suspension of a culture of Staph. aureus was injected into the septum. The strain had been isolated from the infected hand of an intern. The rabbit lived for over two months and was finally destroyed.

Comment.—This result might be said to illustrate that rabbits are not as susceptible to staphylococcic as to pneumococcic infection.

RABBIT 6.—Two-tenths cubic centimeter of the culture from which rabbit 4 was inoculated was injected into the nasal septum. Two days later the left eyelids were closed. In still another day signs of vertigo appeared. There was rotary nystagmus to the left. On the fourth day following injection the labyrinthine disturbance seemed absent or diminished. Seven days after injection, the head was tilted to the left, and the "cranial nystagmus" was to the right. This condition persisted more or less until death, on the thirty-fifth day, except that there was little motion of the head, which was "fixed" in a rotation of 90 degrees to the left—i. e., the top of the head had rotated to the left.

Autopsy showed some thickening of the meninges. Cultures of the latter and of heart blood gave no growth. The lower lobe of the left lung showed gross hyperemia, and microscopic section revealed it to be the site of a low grade exudative inflammation.

Sections of the head revealed an area of cartilaginous necrosis in the septum. Many vessels in the nasal mucosa were engorged. Hemorrhage was present below and also in the olfactory bulbs. The olfactory nerves passing into the septum were normal, and no perineural reaction was noted. No organisms were found in the sections. Sections of the otic labyrinths were rather unsatisfactory for study.

Comment.—This animal had signs of meningitis and labyrinthitis within a few days after inoculation. It appears that the meningitis healed and that it was not the direct cause of death. What the immediate cause of death was is not certain. The pneumonitis might have been a factor. The genesis of the labyrinthitis is not known. Intervening meningitis and sepsis are factors that must be considered.

RABBIT 7.—Two-tenths cubic centimeter of a saline suspension of type 3 pneumococci was injected into the nasal septum. The strain had been isolated from a human case of meningitis. The animal died in less than sixty hours. Autopsy revealed vascular injection of the meninges. Some gross pus was seen in the anterior cranial fossa. On culture this produced a growth of type 3 pneumococci. The heart blood was sterile.

Coronal sections of the head showed unilateral septal ulceration with hemorrhage and fibrinous exudation. Exudation and hemorrhage were present also at the roof of the nose. Organisms were seen in the exudate and in the blood vessels. Some of these vessels adjoined olfactory nerve filaments. No organisms were seen in direct relation to nerve filaments. The surface of the forebrain and the olfactory bulbs were covered with a hemorrhagic and inflammatory exudate. Pneumococci were visible in the hemorrhages.

Comment.—Here again organisms were demonstrated intramucosally and intracranially in dilated blood vessels and hemorrhagic exudates. None were seen in direct relation to the olfactory nerves or the nerve sheaths. However, the blood vessels containing these organisms were in relation to the nerves.

INTRACRANIAL LESIONS CONSEQUENT TO SEPTAL INFECTION

Dural Abscesses.—External abscess secondary to infection of the nasal septum is rare. Benjamins [13] and Unterberger [10] each reported 2 cases. In 3 it followed septal abscess and in 1 submucous resection of the septum. From their records one may conclude that extension of the infection was either by osteitis or by osteomyelitis. One of the patients [13] also had a dural fistula, or pachymeningitis penetrans. Subdural abscess is likewise rare. Benjamins [13] and Berendes [45] each reported 1 case.

Thrombosis of the Dural Sinuses.—Of 11 reported fatal cases of septal abscess, 4 terminated in thrombosis of a cavernous sinus. Only 1 such lesion was found in 27 patients who died subsequent to submucous resection of the nasal septum. Thus it is evident that septal abscess is more prone to extend by thrombophlebitic means than is infection following submucous resection. In Fabricant's case of submucous resection autopsy revealed purulent sphenoethmoiditis as a possible source of the cavernous sinus thrombosis. In Brühl's [53] cases of thrombosis of the superior sagittal sinus the primary focus was the frontal sinus and not the surgically altered nasal septum. Thrombosis of the lateral sinus may follow thrombosis of the cavernous sinus [14] or may be secondary to mastoiditis [17] following submucous resection.

Leptomeningitis.—Purulent meningitis is the most common intracranial suppurative lesion following infection of the nasal septum. Six fatal cases of leptomeningitis following septal abscess are on record. In addition Witchell's [56] case of

56. Witchell, I. S.: Septal Abscess Followed by Cavernous Sinus Thrombosis and Meningitis, M. Rec. **144**:493 (Dec. 2) 1936.

cavernous sinus thrombosis was found to include meningitis. Of 27 patients with fatal intracranial suppuration following submucous resection, 25 died of meningitis. Other cases in which it was not clear whether the meningitis was directly secondary to submucous resection or to sinusitis have been reported.[57] Another such case is reported in this article. Experimental leptomeningitis was demonstrated in several of the rabbits whose protocols have been given.

Abscess of the Brain.—Encephalic cortical abscess following submucous resection of the nasal septum has been reported by Evans.[47] The extension is ascribed to pyemia. An abscess of the frontal lobe of the brain following direct trauma to the septum has been reported by Ertl.[21] A single instance of an abscess of the frontal lobe in a case of septal abscess with autopsy is recorded.[45] Subdural abscess and purulent meningitis were also present.

In reviewing the pathologic intracranial lesions originating in the nasal septum it becomes evident that leptomeningitis is the most common lesion. Thrombosis of the cavernous sinus is next in order and more often follows septal abscess. Localized collections of pus about the dura mater probably represent direct extension by osteitis or osteomyelitis. Abscess of the brain is a rare lesion.

CLINICAL FEATURES AND SURGICAL CONSIDERATIONS

The foregoing pathologic and statistical studies would be of little value if a few clinical factors were not also considered and correlated. Obviously, the discussions which follow are brief since the main purpose of this paper is the study of pathogenesis and of lesions.

Abscess of the Septum.—The clinical diagnosis of septal abscess is usually quite simple. Sometimes a soft septal swelling contains blood (hematoma) instead of pus, but in either case the treatment is the same, namely, incision and drainage. Often a hematoma which has been neglected long enough will suppurate and become an abscess. In the case of Watanabe [11] the septal abscess was most prominent on the side which was subjected to ethmoidectomy (left). This fact raises a question as to unintentional operative trauma of the septum being a factor in starting the abscess. Voorhees [58] recommended that in addition to incision of a septal abscess the cavity should be irrigated, then treated with pure phenol, followed by 95 per cent alcohol, and the walls of the abscess held in apposition with splints for twelve to twenty-four hours. Carter [3] expressed opinions in essential agreement with this view. Menzel [59] does not splint the septal walls until after fever has subsided. Other workers have recommended that drains be inserted in the abscess cavity. Whatever the type of after-care, incision and good drainage are still fundamental in the treatment of septal abscess and the avoidance of complications. To this primary treatment one may now add chemotherapy. Physical therapy in the form of hot and cold applications is still a valuable adjunct to the treatment of septal abscess.

Submucous Resection.—(*a*) Preoperative Considerations: Careful preoperative study is important in preventing complications following operation on the nose. Some patients are prone to rush a doctor into performing an elective operation before a full and proper diagnosis has been made. Sometimes multiple operations are executed in order to save time and money for the patient. Fabricant [14] stressed the importance of proper preoperative examinations and cited the example of his patient who had not been seen for two months, from the time the operation was advised until it was performed. After the fatal illness had set in, he was able to obtain from the parents a history of swimming and of a recent cold. At autopsy purulent sphenoethmoiditis was discovered as a possible cause of the thrombosis of a cavernous sinus which followed submucous resection. Powell's [15] patient likewise had latent suppuration of the sphenoid sinus which produced meningitis following submucous resection. Loeb [1] at the close of his paper gave a long list of "reflections" regarding fatalities following operations on the nose and throat. I commend these to the reader for study and meditation.

(*b*) Surgical Technic: Concerning the large and important subject of the technic of submucous resection little will be said here. Almost needless to say, aseptic technic is necessary, the operative procedure should not go beyond the proper anatomic boundaries, and the operator should respect the fragile nasal structures by exercising gentleness. With rare exceptions submucous resection should be done only with the patient under local anesthesia and preferably in the semirecumbent position.

(*c*) Postoperative Packing: Postoperative packing or splinting is a much disputed subject. Some surgeons never pack the nasal chambers following submucous resection, while others splint the septal flaps tightly. There is little

57. Loeb.[1] Powell.[15]

58. Voorhees, I. W.: Management of Abscess of the Nasal Septum, Laryngoscope **39**:652 (Oct.) 1929.

59. Menzel, K. M.: Zur Behandlung des Septumabszesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:349 (Jan. 7) 1929.

doubt but that tight and prolonged packing predisposes in many cases to postoperative suppurative complications by preventing free drainage. On the other hand, poor splinting may permit free bleeding, and the resulting blood clot may easily suppurate. In Schreyer's [60] report of 9 fatal cases of meningitis following operations on air sinuses there were 3 in which prolonged or repeated packing of the nose could be blamed for the intracranial extension of infection. In 2 of his cases postoperative hemorrhage required packing. Grünwald [61] as long ago as in 1896 definitely stated that postoperative packing of the nose predisposed to intracranial complications. Brühl [53] expressed the belief that in his case of intracranial infection from the frontal sinus following submucous resection the operation and the postoperative packing blocked drainage from the frontal sinus. Halle [55] took issue with him in the specific case, not believing that the septal operation and tamponade predisposed to the fatal issue. Halle apparently uses little packing following submucous resection.

In the instance of Combecher's [9] nonfatal case of reactive meningitis following submucous resection the packing had been left in the nose for "several days" as the patient secretly left the hospital the afternoon following the operation. Removal of the packing and evacuation of a septal abscess resulted in his recovery. In 1 of Hays's [4] fatal cases light nasal pasks were retained for two days after the operation. In case 1 in this report packs were retained for forty-eight hours. Whether retention of packing for forty-eight hours in Kelemen's [49] case of thromboembolism was an important factor is not known, but the fact is recorded. Likewise, what role was played by postoperative packing in Alexander's [62] case is not clear, but the packing may have been a factor in blocking drainage after at least one of the operations, as it was retained for two days. Loeb [1] stated that "two factors combine to influence a meningitis following submucous resection: The perpendicular plate of the ethmoid and the exposure of the medial sides of the septal mucosa with exclusion of drainage brought about by holding the two exposed surfaces together."

Miodowski [20] expressed himself as definitely in favor of good postoperative drainage and as opposed to extensive packing after septal resections. Weinberger,[63] discussing the subject, said, specifically, "It is generally accepted that packing increases the possibility of other complications, notably the infections of the operative field, the sinuses, the ears, the meninges, or the throat." He advocated removing packing in twenty-four hours and held repacking to be dangerous. If a pack is left in for forty-eight hours, the second twenty-four hours favors infection more than the first.

Not only may packing cause intracranial extension of infection from the septum but in connection with submucous resection it may induce otitis media with its potential dangers. Germán [64] reported 6 cases of otitis media following nasal packing and 22 cases of this complication following nasopharyngeal packing. In 1 instance in the latter group abscess of the brain resulted. In 6 of the 22 cases the otitis media followed submucous resection. The detailed mechanism of the extension of infection to the ear will not be discussed in this paper.

While I favor some sort of packing or splinting following submucous resection, I do so with the full realization that too tight packing, prolonged packing, or splinting or packing of any form which impedes free drainage of infected secretions is a menace. There is only one seemingly absolute rule that one must follow. That is never to leave packing in the nose for more than twenty-four hours after an operation on the septum. If there has been a hemorrhage and another hemorrhage is feared if the packing is removed, it is better to take out the packing and renew it immediately if necessary than to leave it in place for forty-eight hours without change.

(*d*) Postoperative Angina: Acute tonsillitis or pharyngitis is not uncommon following submucous resection. In the fatal cases of Errecart [48] and Risch [16] acute tonsillitis developed. In the nonfatal case of extradural abscess and osteomyelitis reported by Benjamins [13] angina developed shortly after surgical intervention. In Loeb's [1] case of fatal otitic meningitis, acute tonsillitis developed after the operation on the septum, and acute purulent otitis media then followed. In Houser's [17] nonfatal case of otitic thrombosis of the lateral sinus the submucous resection was followed by acute tonsillitis in three days and this in turn by acute suppurative otitis media. It seems from these recorded cases that acute tonsillitis following submucous resection is

60. Schreyer, W.: Ueber cerebrale und septische Komplikationen bei Nasennebenhöhleneiterung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:218 (Dec. 18) 1929.

61. Grünwald, L.: Die Lahre von den Naseneiterungen, ed. 2, Munich, J. F. Lehmann, 1896.

62. Alexander, I. H.: Complications Following the Submucous Operation upon the Nasal Septum, New York M. J. **94**:783 (Oct. 14) 1911.

63. Weinberger, N. S.: Submucous Resection—Complications and After-Results, Ann. Otol., Rhin. & Laryng. **32**:387 (June) 1923.

64. Germán, T.: Ueber Otitiden nach Tamponade der Nase und des Nasenrachens ("Tampon-Otitiden"): Bakteriologie und Klinik, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:191 (Sept. 5) 1928.

Collected Cases of Intracranial Suppuration Secondary to Submucous Resection of the Nasal Septum or Septal Abscess

Cases	Author	Sex	Age, Yr.	Primary Septal Condition	Associated Facts of Possible Importance in Pathogenesis	Intracranial Lesion(s)	Probable Mode(s) of Extension	Result	Autopsy
1	Alexander [62]	F	26	Submucous resection	Partial turbinectomy done one week before submucous resection	Leptomeningitis	Not known	Death	No
1	Benjamins [13]	F	12	Septal abscess following coryza	.....	Extradural and subdural abscesses	Osteomyelitis	Death	Yes
1	Benjamins [13]	M	23	Submucous resection	Angina	Extradural abscess	Osteomyelitis	Recovery	
1	Berendes [45]	M	14	Septal abscess	Operation on frontal and ethmoid sinuses because of signs of meningitis	Abscess of frontal lobe of brain, subdural abscess	Said to be by way of "lymphatics"	Death	Yes
1	Brühl [53]	F	16	Submucous resection	Acute frontal sinusitis	Thrombosis of superior sagittal sinus, meningitis	Thrombophlebitis from frontal sinus	Death	Yes
1	Carter [24]	..	..	Septal abscess	.....	Meningitis	Thought to be through cribriform plate	Death	No
1	Carter [24]	..	..	Septal abscess	.....	Meningitis	Thought to be through cribriform plate	Death	No
1	Clay [33]	F	68	Septal abscess (post-traumatic)	.....	Thrombosis of cavernous sinus	Not known	Death	No
1	Combecher [9]	F	17	Septal abscess (post-traumatic)	Influenza 1 month previously	Reactive meningitis	Not known	Recovery	
1	Combecher [9]	F	19	Submucous resection	Septal abscess developed following retention of nasal packing	Reactive meningitis	Not known	Recovery	
1	Cordeiro and Braz Gravina [22]	F	22	Septal abscess (post-traumatic)	.....	Meningitis	Not known	Death	No
3	Dabney [25]	..	..	Submucous resection	.....	Meningitis	Not known	Death	No
1	Dabney [25]	..	..	Submucous resection	Chronic suppurative ethmoiditis	Meningitis	Not known	Death	No
1	Dabney [25]	..	..	Submucous resection	.....	"Cerebrospinal meningitis"	Not known	Death	No
1	Errecart [48]	M	32	Submucous resection	Postoperative angina	Meningitis	Not known	Death	No
1	Erti [21]	..	..	Trauma at root of nose with wood spinter lodged in septum	Operation on frontal and ethmoid sinuses	Meningitis; abscess of frontal lobe	Sheaths of olfactory nerve	Death	Yes
1	Evans [47]	..	..	Submucous resection	.....	Abscess of cerebral cortex	Pyemia	Death	Yes
1	Fabricant [14]	M	33	Submucous resection	Purulent sphenoethmoiditis	Thrombosis of cavernous sinus; localized meningitis	Thrombophlebitis of ethmoid veins	Death	Yes
1	Goldman [32]	..	..	Septal abscess	.....	Thrombosis of cavernous sinus	By veins	Death	?
1	Houser [17]	..	..	Submucous resection	Postoperative tonsillitis, otitis media and mastoiditis	Thrombosis of lateral sinus	Otitic pathway	Recovery	

1	Hays [4]	F	18	Submucous resection	Symptoms of septicemia	Thrombosis of cavernous sinus; meningitis	Not known	Death	No
1	Hofmann [42]	F	4	Septal abscess	.....	Meningitis, erosion of artery at base of brain	Perineural spaces of olfactory nerves	Death	Yes
15	Loeb [1]	..	..	Submucous resection	.....	Meningitis	Not known except that in 2 cases fracture of cribriform plate was blamed	Death	
2	Loeb [1]	..	..	Submucous resection	Bilateral exenteration of ethmoid cells	Meningitis	Not known	Death	
1	Loeb [1]	..	..	Submucous resection	Bilateral exenteration of ethmoid cells	Meningitis	Fracture of cribriform plate	Death	
1	Loeb [1]	..	..	Submucous resection	Intranasal operation on frontal sinuses	Meningitis	Not known	Death	
1	Loeb [1]	..	..	Submucous resection	Ethmoidectomy and other intranasal operations	Meningitis	Not known	Death	
1	Loeb [1]	M	52	Submucous resection	Postoperative tonsillitis and otitis media	Meningitis	Otitic pathway	Death	Yes
2	Miodowski [20]	..	..	Submucous resection	.....	Meningitis	Olfactory perineal spaces	Death	Yes
1	Portmann and Sandler	M	22	Infected septal hematoma	Orbital exploration	Thrombosis of cavernous sinus	Not known	Death	No
1	Powell [15]	M	20	Submucous resection	Sphenoiditis	Meningitis	Not known but thought to be from sphenoid sinuses	Death	Yes
1	Risch [16]	F	30	Submucous resection	Postoperative tonsillitis, otitis media and mastoiditis	Meningitis	Not known but thought to be from sphenoid sinuses	Death	Yes
1	Unterberger [10]	F	3	Septal abscess	Purulent ethmoiditis; bilateral ethmoidectomy	Meningitis, osteomyelitis of cribriform plate with erosion, extradural abscess	Osteomyelitis	Death	Yes
1	Unterberger [10]	M	12	Septal abscess	Orbital abscesses; ethmoidectomy	Meningitis, extradural abscess	Not known but may have been by thrombosis of veins	Recovery	
1	Watanabe [11]	M	18	Septal abscess	Operations on maxillary and ethmoid sinuses two weeks prior to onset of abscess	Meningitis	Said to be by perineural lymphatic spaces	Death	No
1	Wertheim	..	..	Septal abscess	Pansinusitis	Meningitis	Possible extension from frontal sinus	Death	?
1	Witchell [56]	..	..	Traumatic septal abscess	.....	Thrombosis of cavernous sinus, meningitis	Not known	Death	No
1	Rosenvold (this issue)	M	44	Submucous resection	.....	Meningitis	Not known	Death	No
1	Rosenvold (this issue)	M	45	Submucous resection	Bilateral intranasal ethmoidectomy	Meningitis	Not known	Death	No
1	Rosenvold (this issue)	M	25	Crushing of septum	Later external frontal sinusotomy and submucous resection		Not known	Death	No

a complication that may favor intracranial extension of suppuration and should not be regarded lightly. The exact mechanism of extension in those cases in which otitis does not develop as an intermediate infection is not clear. Perhaps the severe tonsillar infection merely represents evidence of weakness or lowers the general resistance of the body to infection. To prevent postoperative tonsillitis it is well not to keep the nose tightly packed for a long time and in cases in which both tonsillectomy and submucous resection are indicated, the former operation preferably precedes the latter by several weeks.

SUMMARY AND CONCLUSIONS

In reviewing the 58 collected cases reported in this study, one is struck with the fact that though the incidence of intracranial suppuration secondary to disease of the nasal septum is low, as might be expected, the mortality associated with this complication is high. It may well be that the sulfonamide drugs will aid in reducing the morbidity and the mortality in the future.

Extension of infection from the septum to the intradural structures may be by direct trauma (usually operative), osteomyelitis or erosion of bone, thrombosis of intervening venous channels, and by way of the olfactory perineural sheaths. The latter two modes are especially important if the extension is not due to direct perforative trauma. Extension to the intradural structures by way of the ordinary peripheral lymphatics probably does not occur. Turner and Reynolds [41] denied the existence of any connection between the peripheral lymphatics and the intradural structures. Animal experiments conducted in this study favored a vascular (thrombophlebitic) type of spread from the septum to the intradural contents. It is possible that the mode of inoculating the animals may have affected the final results.

Meningitis is the most common lesion secondary to either submucous resection of the nasal septum or septal abscess. Thrombosis of the cavernous sinus occurs more commonly following septal abscess. Localized collections of pus, such as a dural abscess or an abscess of the brain, are rare complications of septal infection.

Improper preoperative study and care, faulty operative technics and poor postoperative management account for certain fatalities following submucous resection. Multiple unrelated operations are to be condemned in principle as is a series of operations without proper intervals of time in which the patient may recuperate properly. When submucous resection and operations on sinuses are performed simultaneously, it may be difficult to trace the pathways of extension and thereby to treat the complications wisely. Acute tonsillitis following submucous resection is a serious omen of impending complications.

It is hoped that whenever a fatality occurs following septal abscess, septal trauma or submucous resection, the attending physicians will take the time to report the case. Among the 56 cases included in this survey there were 32 in which for all practical purposes it can be said the pathway of extension was not known. In the remaining 24 instances a reasonable hypothesis was available to explain the pathogenesis. (The 2 cases in which extension to the intracranial structures was by way of otitis media which followed submucous resection are not included in these figures.) Among the 52 fatal cases there were only 11 in which an autopsy was recorded. Until more reports are at hand, studies such as this one will be handicapped and progress in the prevention and treatment of intracranial complications will not be as rapid as it should be.

1687 Hill Drive (Eagle Rock), Los Angeles 41.

CHEMOTHERAPY AND BIOTHERAPY

THEIR RELATION TO THE PREVENTION AND TREATMENT OF DISEASES OF THE EAR, NOSE AND THROAT

JOHN A. KOLMER, M.D.

Professor of Medicine, Temple University School of Medicine; Director of the Research Institute of Cutaneous Medicine

PHILADELPHIA

Because the route of infection of many of the pathogenic bacteria and filtrable viruses is by way of the upper respiratory tract, biotherapy and chemotherapy in the prevention and treatment of the local and systemic diseases they produce are of particular interest and importance to otolaryngologists, especially those practicing in the temperate and colder climates, where droplet infections are of frequent occurrence. It is true that many of the infectious diseases of the ears, nose and throat remain localized, but not infrequently they involve by extension the lower respiratory tract with production of bronchitis, pneumonia and bronchiectasis, the meninges with production of severe and highly fatal types of suppurative meningitis, and the blood with production of septicemia. Furthermore, chronic localized areas of infection, especially those in the nasal accessory sinuses and tonsils, are frequently important in relation to the etiology of the diseases due to focal infection.

PROPHYLACTIC VACCINATION

Pertussis.—There are a few diseases in which otolaryngologists cannot escape a special interest and responsibility from the standpoint of prophylactic vaccination. Although pertussis does not ordinarily come within this field, yet some of its complications, with special reference to otitis media, mastoiditis and sinusitis, may require the otolaryngologists' diagnostic skill and therapeutic management. As shown by Dauer,[1] if the mortality rate, which has declined spontaneously by 80 per cent in the past fifteen years, continues to decline so rapidly, it may not be possible to prove statistically the value of prophylactic immunization, but I believe that this has been shown to be sufficiently safe and effective to justify otolaryngologists in encouraging parents to have their children immunized six to nine months after birth and certainly before 2 years of age. It is true that about 40 per cent of deaths occur in infants under 6 months of age and that the effectiveness of active immunization in this age group is as yet unknown, but in a large percentage of older children it apparently engenders sufficient immunity to afford protection over the age of great susceptibility, with chances of the immunity being subsequently prolonged through supplementary immunization by mild or clinically unrecognized attacks of the disease. According to Cohen and Scadron,[2] however, it is possible to immunize newborn babies by vaccinating pregnant women with pertussis vaccine during the last trimester. For this purpose the authors advise that a total dose of 150 billion organisms be given in six injections at two week intervals.

Diphtheria and Scarlet Fever.—The same argument may be advocated for prophylaxis against diphtheria. Immunization with vaccines of plain or alum-precipitated toxoids is so safe and so highly effective that all children should be immunized between 1 and 2 years of age. At 5 to 6 years of age the immunized child should be given the Schick test, and if positive reactions are observed, should be reimmunized.

Scarlet fever is another disease in which otolaryngologists cannot escape responsibility in routinely recommending prophylactic immunization of children between 2 and 4 years of age as a safe and effective means of protecting the majority over the age of greatest susceptibility, with chances of prolongation of effective immunity through immunization by clinically mild and unrecognized infections with beta hemolytic streptococci.

Common Cold.—Undoubtedly, however, the possibility of effective vaccination against the infectious common cold, so aptly designated by

This article is an address delivered, by invitation, before the New England Oto-Laryngological Society, in Boston, Feb. 16, 1944.

1. Dauer, C. C.: Reported Whooping Cough Morbidity and Mortality in the United States, Pub. Health Rep. **58**:661-672 (April 23) 1943.

2. Cohen, P., and Scadron, S. J.: The Placental Transmission of Protective Antibodies Against Whooping Cough, J. A. M. A. **121**:656-661 (Feb. 27) 1943.

Miller [3] as "public malady number one," is of particular interest to otolaryngologists and to all other physicians and surgeons as well. While this disease is now regarded as being due to a filtrable virus, the latter has not been successfully cultivated. Consequently, there is no method available at the present time for active immunization with vaccines prepared of it. But even were it otherwise one would wonder whether effective immunity could be engendered by vaccination with virus in view of the fact that the resistance acquired from repeated attacks of the disease is apparently of brief duration. In other words, it appears that the virus either is low in antigenic activity or exists in many immunologically specific strains so that recovery from infection with any of them still leaves the patients susceptible to others.

Apparently, however, infection with this virus produces only a relatively mild or trivial cold of but a few days' duration. This usually permits its victims to be up and about, with transmission of the infection to others, which, along with the fact that the virus is highly contagious and the period of acquired immunity brief, readily accounts for its designation as the "common cold." Prolongation of the disease and the production of its complications, such as acute and chronic sinusitis, laryngitis, bronchitis and labyrinthitis, are undoubtedly due to secondary bacterial infection with streptococci, staphylococci, pneumococci or other micro-organisms occurring when the local resistance to such microbes has been lowered or their virulence enhanced by the primary viral infection. Vaccination against the infectious common cold, therefore, is concerned at present only with the possibility of effective immunization against this important secondary bacterial infection. That this is highly desirable, no one can deny; that it can be successfully achieved is both debatable and controversial. Consequently, few subjects in prophylactic immunization are more difficult to evaluate.

In the first place, a goodly percentage of the laity who believe themselves victims of repeated attacks of the infectious common cold may not have this viral disease at all, since the signs and symptoms of mild influenza or grip, vasomotor rhinitis, primary bacterial infections of the upper respiratory tract and especially acute exacerbations of chronic sinusitis are so readily mistaken for those of the common cold. Indeed, physicians, including even otolaryngologists, are not always as careful as they should be in selecting patients for active immunization. For this reason reports on the results of mass immunization of large groups of improperly selected persons are readily misleading and of limited scientific value.

In the second place, it is a reasonable assumption that a large percentage of the laity requesting vaccination against the disease are likely to be those who exercise more than ordinary care in avoiding contact with it, and this along with other preventive measures of hygienic value may readily enough account for the freedom from attacks that is so likely to be credited to successful immunization.

Nevertheless, my experience during the past twenty-five years has given me a favorable impression of the clinical value of active immunization against the secondary bacterial infection of the disease in properly selected patients. In this connection I should add that I never consent to the procedure until the patient has been carefully examined by an otolaryngologist for chronic sinusitis, polyps, a deviated nasal septum and vasomotor rhinitis due to allergy to foods or airborne allergens; the presence of any of these conditions is regarded as a contraindication. Furthermore, my experience has been limited almost entirely to the administration of carefully prepared autogenous vaccines. My practice is to wait until the first infectious common cold has occurred; then, on or about the fifth day, cultures are made on enriched mediums for the cultivation of streptococci, pneumococci, Haemophilus influenzae and other bacteria. The micro-organisms isolated, including staphylococci, are then cultivated in broth mediums for at least five days to permit the production of exotoxins, since these possess the maximum of vaccinogenic activity. Equal parts of the broth cultures of selected micro-organisms, with special reference to staphylococci, streptococci and pneumococci, are then mixed, diluted with sterile saline solution so that the mixture will contain approximately a total of 1,000 million micro-organisms per cubic centimeter, sterilized by the addition of phenol in 0.5 per cent concentration and left in an incubator at 37 C. for at least twenty-four hours, when tests for sterility are made. To adults these vaccines are given in doses of 0.2, 0.4, 0.6, 0.8 and 1.0 cc. by subcutaneous injection at weekly intervals, followed by monthly injections of 1 cc. during the autumn, winter and spring months of the year. Immunized persons may continue to have mild attacks of the disease lasting for one to three or four days and ascribed to infection with the virus, but at least 60 to 70 per cent apparently escape prolonged attacks and complications due to the secondary bacterial infection. It is my impression that the immunity

3. Miller, L. M.: Public Malady Number One, Hygeia **18**:8 (Jan.) 1940.

is not usually of more than one year's duration, but I have reasons for surmising that in some persons it has endured over a period of several years.

Many physicians have been favorably impressed with the subcutaneous administration of highly polyvalent stock vaccines, but since the etiologic agents of the secondary infections are likely to vary according to locality, and since it is now known that staphylococci, streptococci and pneumococci exist in serologically specific strains, it would appear that autogenous vaccines are to be preferred. It is to be admitted, however, that a properly prepared stock vaccine is likely to be more effective than a poorly prepared autogenous one.

As to oral administration of polyvalent desiccated bacterial vaccines, my experience is too limited to permit an expression of opinion on its prophylactic value. I have the distinct impression, however, that results in individual cases in private practice are more encouraging than is indicated by the reports on the mass immunizations carried on by various industrial organizations and insurance companies. Whether this is due to the fortuitous circumstances previously mentioned or to actual immunity cannot be stated. For my own part, I am willing to leave the matter open until thoroughly conducted investigations are made on properly selected subjects with adequate controls, including serologic studies bearing on the production of antibodies before, during and after immunization.

Influenza.—Epidemic influenza, including its mild form grip, is now known to be due to a filtrable virus which is believed to exist in at least two immunologic specific strains designated as A and B, with variants of each. In other words, the disease is probably due to a group of closely related viruses. Fortunately, strains of group A can be readily cultivated, and various investigators [4] have reported encouraging results in the active immunization of small groups of human beings with vaccines prepared from them, but the matter is still in the experimental stage. In this connection it may be stated, however, that the viruses of influenza are apparently similar to the virus of the infectious common cold in the sense of reducing resistance to secondary bacterial infections, with special reference to hemolytic streptococci of group A, which are largely, if not entirely, responsible for the severity and mortality of the disease, particularly through the production of atypical pneumonias.

4. Horsfal, F. L., Jr.; Lennette, E. H.; Rickard, E. R., and Hirst, G. K.: Studies on the Efficacy of a Complex Vaccine Against Influenza A, Pub. Health Rep. **56**:1863-1875 (Sept.) 1941. Henle, W.; Henle, G., and Stokes, J., Jr.: Demonstration of the Efficacy of Vaccination Against Influenza Type A by Experimental Infection of Human Beings, J. Immunol. **46**:163-175 (March) 1943.

VACCINE THERAPY

The efficacy of vaccines in the treatment of diseases of the ear, nose and throat is likewise difficult to evaluate in spite of the accumulation of a large literature on the subject. My own experience, which has been extensive, has been based entirely on the preparation and administration of autogenous vaccines. Of one thing I am certain, namely, that the purpose of treatment with autogenous vaccines is frequently defeated and the therapy rendered almost useless by carelessness on the part of some otolaryngologists and other physicians in makng the cultures. Regardless of how skilfully bacteriologic examinations and vaccines are made, specific therapeutic results cannot be expected unless the micro-organisms actually and primarily responsible for the infections are utilized. Altogether too frequently cultures are prepared on plain agar or other mediums inadequate for the cultivation and isolation of hemolytic and other types of streptococci, pneumococci, Friedländer's bacillus, H. influenzae and others likely to be of primary importance. Consequently, due care should be exercised in the selection of appropriate enriched mediums and in the securing of exudates from actual sites of infection, as free as possible from contamination, for bacteriologic diagnosis and the preparation of vaccines.

Under these conditions I believe that the prolonged administration of autogenous vaccines, prepared as previously described in order to incorporate any exogenous toxins that may be produced by streptococci and staphylococci, is sometimes distinctly helpful and worth while as an adjuvant in the treatment of chronic sinusitis and chronic otitis media, provided that adequate surgical drainage and appropriate local measures are employed at the same time. Otherwise, experience has taught me that vaccine therapy alone is likely to prove worthless and disappointing. I have never employed stock vaccines for fear that they may not contain micro-organisms corresponding in serologic specificity to those producing the infection. Furthermore, I believe that vaccines tend to undergo some deterioration in antigenic activity on aging, especially if kept at room temperature. For these reasons it appears to me that freshly prepared autogenous vaccines are to be preferred, although it is likely that well prepared stock ones may be more efficacious than poorly prepared autogenous vaccines. I recommend that the former be administered at intervals of five

to seven days and the dosage arranged to avoid excessive focal reactions. Intracutaneous injections are likely to prove more effective than subcutaneous ones but may be objected to because of local reactions at the sites of injection. When vaccines carry a total of about 1,000 million micro-organisms per cubic centimeter, I believe that the first dose for adults may be 0.1 or 0.2 cc., to be given by subcutanous injection. But no laboratory should attempt to make out a dosage scale for subsequent injections. This should be determined only by the physician on the basis of the focal and general reactions following each injection. For intracutaneous injection the dose is usually ten times less than that for subcutaneous injection.

I am convinced that properly prepared and properly administered autogenous vaccines are of great value in the treatment of chronic bronchitis and infective asthma, which are likely, if not almost invariably, due to infection of the bronchi through postnasal dripping from a chronically infected upper respiratory tract, with special reference to ethmoiditis and sphenoiditis. Here again vaccination is only an adjuvant and not usually a short cut or royal road to control by being a substitute for surgical drainage and other appropriate local measures.

As a clinician I am also an ardent believer in focal infection as a frequent cause of systemic disease, even though many considerations relative to its mechanism are still uncertain and controversial. Consequently, I frequently turn to otolaryngologists for surgical drainage or extirpation of primary foci of infection in the upper respiratory tract and always include the administration of a carefully prepared autogenous vaccine in the follow-up therapeutic program. In my opinion the disposal of surgically removed tonsils before cultures have been made and an autogenous vaccine prepared is a real sin of omission in the treatment of diseases of focal infections believed or assumed to be in etiologic relation to chronic tonsillitis. It is true that the drainage or the extirpation of obvious primary foci of infection alone may be therapeutically disappointing in regard to the treatment of the secondary lesions, since it may be a matter of "locking the stable after the horse has been stolen," but I believe that such measures along with autogenous vaccine therapy are right, proper and frequently helpful.

SERUM THERAPY

It may be mentioned that diphtheria antitoxin is still the only specific agent available for the immediate prophylaxis and treatment of diphtheria since the sulfonamide compounds have proved ineffective, although these are sometimes indicated as adjuvants for combating severe secondary infections with hemolytic streptococci, pneumococci or Staphylococcus aureus.

Sulfonamide therapy alone, however, may fail in the treatment of streptococcic, pneumococcic or influenzal meningitis or that due to Friedländer's bacillus, frequently secondary to suppurative disease of the mastoid and nasal accessory sinuses. Consequently I believe that it is advisable always to institute promptly the best possible surgical drainage of the primary focus of infection and, in cases of pneumococcic or influenzal meningitis, to administer type-specific immune serum in adequate amounts in addition to a sulfonamide compound in maximum doses. At least there appears to be a synergistic action of therapeutic value between the two agents in the sense that the specific antibody supplied by the immune serum materially aids in the disposal, through phagocytosis, of those infecting micro-organisms injured by the sulfonamide compound. There is still some difference of opinion regarding the route of choice for the administration of serum but I administer it intravenously once a day and intrathecally twice a day in all cases. At least I have never seen any harm attending intrathecal injections when these have been made by the gravity method following spinal drainages, and I believe that they facilitate phagocytosis of the micro-organisms.

In this connection a relatively simple precipitin test has proved helpful as a guide in serum therapy. It is conducted in a small test tube by carefully overlaying 0.1 to 0.2 cc. of type-specific antipneumococcic serum with 1 cc. of spinal fluid clarified by centrifugation. After this preparation has stood about fifteen minutes at room temperature, a ring of precipitation at the line of contact indicates that there is present in the spinal fluid a free type-specific soluble polysaccharide and that there is need for the administration of additional serum. Absence of this reaction may be accepted as an indication that serum therapy may be stopped or the dose reduced. In the case of influenzal meningitis, a similar test is conducted with anti-influenzal serum type B.

While immune serum for the treatment of meningitis due to Friedländer's bacillus type A is worthy of trial, its value cannot be stated at the present time. Polyvalent antistreptococcus serum has been used extensively but with disappointing results; monovalent immune serums for the specific types of beta hemolytic streptococci of group A should be employed when available. Otherwise, in cases of meningitis due to hemolytic streptococcus I frequently attempt

to supply antibody for these micro-organisms by the administration of serum or plasma (or by the transfusion of compatible blood) from donors convalescent after scarlet fever.

THERAPY WITH SULFONAMIDE COMPOUNDS

The discovery of the wide range of therapeutic effectiveness of the sulfonamide compounds in the treatment of the various local and systemic diseases due to pathogenic bacteria constitutes one of the most remarkable chapters in the history of therapeutic science and outranks in importance the discovery of arsphenamine. So far as otolaryngology is concerned, this is particularly true in relation to infections due to aerobic hemolytic streptococci of group A, pneumococci, meningococci and, to a lesser extent, staphylococci. Unfortunately, however, chemotherapy has not proved equally effective in the treatment of infections due to aerobic hemolytic streptococci of group B, anaerobic streptococci, nonhemolytic streptococci, streptococci of the viridans type, Friedländer's bacillus, Bacillus pyocyaneus, H. influenzae and Corynebacterium diphtheriae, all frequently of importance in relation to diseases of the ears, nose and throat. Time does not permit me to review the large literature which has accumulated on the local, enteral or parenteral administration of these compounds in relation to otolaryngology. But, even if it were otherwise, I should hesitate to pass critical judgment on it since I am not an otolaryngologist. As a chemotherapeutist, however, I may be permitted to pass in brief review what I believe are some of the important principles involved, along with their practical application.

Principles.—In the first place, the indiscriminate use of the sulfonamide compounds is to be regretted. This judgment is based not only on the possibility and the probability of various pathogenic bacteria acquiring resistance to the sulfonamide compounds through the frequent use of these compounds in the treatment of minor infections but also on the fact that such use breaks one of the cardinal principles in therapeutics. I realize, however, that it is difficult for otolaryngologists and other physicians to refrain from using these compounds in the treatment of the secondary bacterial infections of the common cold, acute exacerbations of chronic infections of the nasal accessory sinuses, acute pharyngitis, acute laryngitis and the like. Indeed, I frequently use them myself in these conditions and so far have had no regret for doing so. Sulfadiazine is my choice, in an initial dose of 1 Gm. for adults followed by the same dose every eight hours over a period of three or four days. Spink [5] stated that chemotherapy should not be used for this group of infections except in (*a*) patients with severe infections actually caused by hemolytic streptococci, (*b*) patients with colds who are known to have cardiac defects, to forestall bacterial endocarditis, and (*c*) obstetric patients who have contracted infection of the respiratory tract at or near term.

In the second place, treatment with the sulfonamide compounds is not always a short cut or royal road to control of disease. This is particularly true in reference to severe infections. In other words, clinical experience has shown that the otolaryngologist cannot afford to omit anything else of proved value in treatment, with special reference to prompt and adequate surgical measures when resort to these is possible, especially drainage of pus and removal of necrotic bone and infected thrombi in the mastoid processes, frontal bones or calvarium, as emphasized by Porter [6] and others with respect to the treatment of osteomyelitis or of thrombosis of a lateral sinus. In regard to thrombophlebitis and especially thrombosis of a cavernous sinus Porter has also recommended the administration of heparin for the prevention of clot formation while sulfadiazine is being given. The same recommendation applies to the simultaneous use of blood transfusions and those immune serums of known therapeutic value. Indeed, since it appears that the effectiveness of the sulfonamide compounds is mainly due to their crippling effects on the proliferative activities of bacteria, making easier the final disposal of the latter by immunologic mechanisms and especially by phagocytosis on the part of the cells of the reticuloendothelial system, aided by antitoxins, opsonins and agglutinins, I am always in favor of using appropriate immune serums for furnishing the hard-pushed patient with these antibodies, especially in the treatment of septicemia or acute suppurative meningitis. Furthermore, it is not unlikely that the simultaneous administration of well prepared autogenous vaccines based on skilful bacteriologic examinations for the purpose of securing the organism or organisms of primary infection or, otherwise, of well prepared stock vaccines may be helpful in the treatment of some of the bacterial diseases, especially those of the ears, nose and throat, including the nasal accessory sinuses, if it is true, as I believe it to be, that specific antibodies may increase the therapeutic effectiveness of the sulfonamide compounds.

5. Spink, W. W.: The Use and Abuse of Chemotherapy, Minnesota Med. **24**:988-990 (Dec.) 1942.

6. Porter, C. T.: Chemotherapy from the Standpoint of the Otolaryngologist, Tr. Am. Acad. Ophth. **47**:34-35 (Sept.-Oct.) 1942.

In the third place, due emphasis should be placed on early treatment and on administration of adequate doses. Here again there is a close analogy between chemotherapy and serum therapy. For example, clinicians have long since learned that the earlier adequate amounts of antitoxin are administered in diphtheria, the better the prognosis. The same observation may be made as to sulfonamide therapy, not to mention that the sooner appropriate treatment for early syphilis is instituted the greater the chances of complete recovery. It is true that the sulfonamide compounds are not without possible toxic effects due to their acetylation or conjugation, but after all the great majority of the latter are of a minor character and in general terms the sulfonamide compounds are of remarkably low toxicity. In other words, delay in treatment and timidity in dosage are not infrequently the primary causes of failure of sulfonamide therapy, especially in the treatment of severe and dangerous infections.

Whenever possible, oral administration is probably always to be preferred, with the administration of double the usual amount for the first one or several doses. Otherwise, parenteral administration is possible with most of the sulfonamide compounds whenever necessary or advisable. Thus 0.8 per cent solutions of sulfanilamide may be given subcutaneously, intravenously or intrathecally, azosulfamide [7] intramuscularly, or the soluble sodium salts of sulfathiazole, sulfapyridine or sulfadiazine intravenously. Fortunately, 0.8 per cent solutions of the soluble sodium sulfadiazine can be given intramuscularly without excessive local reaction and with prompt absorption; this route of administration is particularly serviceable when the compound is administered to children in the treatment of meningococcic, pneumococcic, streptococcic and influenzal meningitis. Not infrequently a combination of oral and parenteral administration is advisable, as likewise a combination of oral and local administration, to which further reference will be made.

Fortunately, the dosages of all of the sulfonamide compounds are the same. Under the conditions, relatively simple rules suffice for their administration, subject to change in different diseases and individual cases. In the treatment of severe infections, with special reference to septicemia and meningitis, I usually calculate the total dose for twenty-four hours on the basis of 1 or 2 grains (0.06 or 0.13 Gm.) per pound of body weight. This total is divided into six parts, with a dose being given every four hours, day and night, by oral or parenteral administration or a combination of the two. For less severe infections the total amount per twenty-four hours is based on ½ grain (0.03 Gm.) per pound of weight; it is divided into six parts, with a dose being given every four hours, or into four parts, with a dose being given every six hours. The total dose in cases of minor infections and especially those of a chronic character is usually based on ¼ grain (0.016 Gm.) per pound of weight; it is divided into four parts, with a dose being given every six hours. In most instances I usually administer at least 5 to 10 grains (0.32 to 0.65 Gm.) of sodium bicarbonate with each dose in an effort to prevent nausea and vomiting due to acidosis. In regard to febrile patients, I believe that the compounds should be administered until the patients are fever free for at least seventy-two hours before doses are reduced.

The cyanosis that occurs when large doses are given is of harmless character, and no attention need be paid to it, but one should warn the patient or the family of its probable occurrence. All ambulatory patients should be warned of the possible occurrence of disorientation and dizziness, especially in relation to the hazards of driving an automobile. Needless to state, the blood should be examined at frequent intervals for possible hemolytic anemia and leukopenia when large doses are administered. The former is not dangerous, but if the latter is ignored or overlooked it may progress to agranulocytosis, which constitutes one of the gravest of complications in relation to chemotherapy.

Natural allergy to a sulfonamide compound occurs but is fortunately uncommon. It is usually expressed as high fever with cutaneous rashes following the first dose. Acquired allergy is much more frequent and in my opinion is usually responsible for the drug fever sometimes occurring about ten days or longer after continuous treatment with a sulfonamide compound. It should never be ignored, as it is sometimes the forerunner of the dreadful agranulocytosis. Fortunately, a patient allergic to one sulfonamide compound may not be allergic to another in case a continuation of treatment is necessary or advisable. A simple and safe test is the oral administration of 5 grains with hourly reading of temperature over a period of at least four to six hours.

It is also advisable to examine the urine for blood when large doses are being administered. Hematuria is usually due to irritation of the uriniferous tubules by crystals of the conjugated or the acetylated compound. The gross appearance of the urine may be insufficient for the

7. Azosulfamide is disodium 4-sulfamidophenyl-2′-azo, 7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfate.

detection of hematuria, which may be present and discoverable only by microscopic examination. In this connection it is to be remembered, however, that the sediment obtained by centrifuging 15 cc. of normal urine frequently contains from 2 to 4 erythrocytes per microscopic field so that, contrary to widespread impression, the presence of a few erythrocytes in patients under treatment with sulfonamide compounds does not necessarily constitute microscopic hematuria.

Unfortunately, not all human beings absorb the sulfonamide compounds equally well from the gastrointestinal tract. Consequently, determinations of the concentration of the free compound in the blood should be made at intervals, especially in cases of severe infection, to insure adequate concentration by alteration of dosage or of method of administration. Such determinations are not always easily made in private practice but when necessary at least one should be made after the first twenty-four hours of treatment, the blood being taken midway between any two doses. With ambulatory patients, however, such determinations may be safely omitted unless there are reasons for suspecting deficient absorption. In view of the importance of the concentration of free compound in the blood I lay great emphasis on making sure that the patient or those responsible for him understand the importance of taking the sulfonamide compound day and night at exactly the intervals prescribed by the physician.

Bacteriologic Diagnosis in Relation to Chemotherapy.—As previously stated, indiscriminate use of sulfonamide compounds is inadvisable. Indeed I believe that the use and especially the choice, of such a compound should be based on bacteriologic diagnosis more frequently than they commonly are. No one can appreciate the possible mistakes in bacteriologic diagnosis more than I and especially when it is remembered that no examination can be better than the laboratory conducting it. In this connection, however, it is only right and proper to point out that errors are not infrequently due primarily to the methods of the physician or the surgeon making the smear or the culture. Unless a proper technic is employed in securing the organism or organisms responsible for the infection, the purpose of the examination is defeated at the outset, because a laboratory can report only what it finds instead of what the physician expects or desires. For example, such organisms as streptococci, pneumococci and H. influenzae are not at all likely to grow on plain agar, with the result that only staphylococci, diphtheroid bacilli, B. pyocyaneus and the like are apt to be found when this medium is used. In passing, it is also worthy of emphasis that only positive blood cultures that have been made with all precautions aganst contamination are acceptable for establishing the diagnosis of septicemia. Indeed, a severe local infection without septicemia may present all of the clinical manifestations of the latter, on the one hand, while, on the other, a positive blood culture may be a matter of surprise in a case of a minor primary infection of tissue.

Unfortunately, normal bacterial flora can be confusing in the interpretation of the laboratory report and especially in the examination of the nose and throat. Cultures of material from the normal nose and throat will invariably show various types of staphylococci and streptococci and quite frequently pneumococci, Micrococcus catarrhalis and other gram-negative diplococci, diphtheroid bacilli, H. influenzae and Friedländer's bacillus. Consequently I maintain that no bacteriologic examination alone can be the basis for a decision on whether or not there is sufficient infection of the tonsils to indicate or warrant their surgical removal. Nor do smears showing many spirochetes and fusiform bacilli necessarily indicate that Vincent's angina alone is present, since those bacilli may be only a secondary and relatively unimportant secondary infection in a patient with agranulocytosis, on the one hand, or a patient with leukemia, on the other. Indeed, I believe that the otolaryngologist should seldom accept a bacteriologic diagnosis of Vincent's angina until at least a total leukocyte count has been made.

Under the circumstances the otolaryngologist should make smears and cultures as carefully as possible and, if necessary, under the guidance or the advice of a bacteriologist. The final interpretation, however, of all laboratory examinations, including the serologic tests for syphilis, is the function and duty of the clinician. Acute infections are usually due to a single strain of organisms, whereas chronic infections are usually due to two or more strains. Once a decision is reached, I believe the choice of a sulfonamide compound for treatment may be in order of preference as follows:

Staphylococcic infections: sulfathiazole, sulfadiazine
Hemolytic streptococcus infections: sulfadiazine, sulfanilamide, azosulfamide
Streptococcus viridans infections: sulfapyridine
Pneumococcic infections: sulfadiazine
Meningococcic infections: sulfadiazine, sulfanilamide
Infections with Friedländer's bacillus: sulfadiazine, sulfapyridine
B. pyocyaneus infections: sulfanilamide, sulfathiazole
H. influenzae infections: sulfadiazine, sulfanilamide

It will be observed that the sulfonamide compounds have a wide range of therapeutic activity in bacterial diseases. Consequently they should

be of use in the treatment of the following diseases of the ears, nose and throat: erysipelas, furunculosis, acute and chronic otitis media, acute and chronic sinusitis, acute tonsillitis and peritonsillar abscess, acute septic sore throat, acute and chronic pharyngitis and meningitis due to infection with streptococci, pneumococci, Friedländer's bacillus and H. influenzae. Indeed, I believe that they are likewise of use in the treatment of the acute infectious common cold, not because they are effective against infection with the virus but because they combat the secondary bacterial infections which inevitably follow owing to lowered resistance and which probably are alone responsible for infections of the nasal accessory sinuses and the lower respiratory tract. Unfortunately, the sulfonamide compounds are ineffective in the treatment of diphtheria except when a severe secondary infection with Staph. aureus, hemolytic streptococci or pneumococci is present; in the latter case they may be employed along with antitoxin. Furthermore, I commonly employ them in the treatment of scarlet fever with the hope of reducing the incidence of otitis media and mastoiditis, excessive cervical adenitis and glomerulonephritis; likewise, in the treatment of pertussis and measles, not with the hope of influencing the primary infections due to the causative viruses but with the hope and expectation of preventing secondary bacterial infections of the ears, mastoid cells and lungs. But whether or not the oral administration of these compounds in the prophylaxis and treatment of the less dangerous and minor diseases is justifiable must rest on the judgment of the individual physician or surgeon.

Local Chemotherapy.—Since the local disinfection of tissues by the topical application of chemical agents is just as much a part of chemotherapy as the specific treatment of syphilis with arsphenamine, chemotherapy in this sense has long played an important role in the treatment of many diseases of the ears, nose and throat due to local infections. Until the introduction of the sulfonamide compounds, however, the local application of the silver and mercury salts, the aniline dyes and other chemical compounds was of limited therapeutic value. No wonder, therefore, that otolaryngologists turned hopefully to the local application of sulfonamide compounds especially in the treatment of chronic infection of the nasal accessory sinuses, otitis media and postoperative wounds. They have made local applications of 0.8 per cent aqueous solution of sulfanilamide, 5 per cent solution of azosulfamide, 2.5 per cent solution of sulfadiazine in triethanolamine-technical, 2.5 per cent solution of sodium sulfathiazole in dl-desoxyephedrine hydrochloride, a 5 per cent suspension of the microcrystals of sulfathiazole in saline solution or isotonic 1 per cent solution of paredrine hydrobromide (p-hydroxy-α-methylphenylethylamine hydrobromide), ointments or jellies containing sulfathiazole in concentrations of 5 to 25 per cent of insufflated finely divided powders of sulfanilamide, sulfathiazole or sulfadiazine. Regarding the clinical value of such applications there is still considerable difference of opinion although the results have been generally sufficiently encouraging to warrant continued trial in selected cases. Certainly the medicament employed should be nontoxic, compatible with ciliary activity, possessed of a physiologic nasal p_H, nontraumatizing to the mucous membrane and without destructive effect on leukocytes, which are so important in phagocytosis. I have been more interested in the finely divided powders and the suspensions than in the solutions because of the possibility and the probability of their exerting more prolonged local antibacterial activity. And since many chronic purulent diseases of the rhino-otolaryngeal tract are apt to be due to mixed bacterial infections, I have suggested using a mixture of equal parts of the powders of sulfanilamide, sulfathiazole and sulfadiazine of such fineness as to pass a 40 to 100 mesh.[8]

However, owing to the ease and frequency with which disinfectants may be introduced into the conjunctival sacs, the chemotherapy of conjunctivitis by local disinfection always has been and probably will continue to be more successful than chemotherapy of the ears and nose, with special reference to the nasal accessory sinuses, unless otolaryngologists succeed in working out methods whereby patients can apply local disinfectants as frequently and thoroughly as conditions demand. No wonder that local disinfection with solutions of chemical agents has so largely failed in the treatment of chronic sinusitis when it is remembered that one or two treatments per week mean that the contact between a disinfectant and the infected tissues is probably to be measured in minutes so far as bactericidal concentrations are concerned. Indeed, the high incidence of chronic sinusitis, the frequency with which it is subject to exacerbations with complications and the tendency for it to produce chronic bronchitis with bronchiectasis, as well as the frequency with which it is in etiologic relationship to the diseases due to focal infection, render it one of the most important problems

8. Kolmer, J. A.: Chemotherapy in Relation to Ophthalmology and Otolaryngology with Special Reference to the Sulfonamide Compounds, Tr. Am. Acad. Ophth. **47**:36-45 (Sept.-Oct.) 1942.

in treatment, readily meriting and demanding the best efforts of otolaryngologists in the solution.

Prophylactic Use of Sulfonamide Compounds.—Furthermore, since introducing crystals of sulfanilamide or sulfacetimide (acetylsulfanilamide) into wounds and the peritoneal cavity has proved so successful in the prevention of infection, otolaryngologists do well to consider carefully whether the local application or the oral administration of sulfonamide compounds may not be prophylactic against many of the diseases due to infection of wounds and cavities. For example, if clinical experience has indicated the types of cases of mastoiditis and sinusitis most likely to result in extension of hemolytic streptococcus or pneumococcus infection to the meninges after operations, it seems only sensible and reasonable to try to protect the patient against these dreadful complications by starting the administration of a sulfonamide compound before and certainly immediately after operation. Indeed, the possible prevention of acute suppurative mastoiditis as a complication of otitis media by prompt administration of a sulfonamide compound, chosen on the basis of the results of a bacteriologic examination, also appears advisable although it is true that it may mask mastoiditis. The latter, however, should be detectable in most cases by a skilful roentgenologic examination. In this connection it also seems advisable to insufflate a powdered sulfonamide compound on the tissues after submucous resection, ethmoidectomy or other intranasal operation and tonsillectomy not only because it may be helpful in the prevention of capillary oozing but especially because it may control exacerbations of infection or the development of secondary infections. In some instances I believe this local disinfection should be combined with the oral administration of a sulfonamide compound for at least three to five days. For adults the oral dose may be 15 grains (0.1 Gm.) every six to eight hours day and night; for children the dose would be less, according to body weight.

PENICILLIN, TYROTHRICIN AND PATULIN THERAPY

Until more data are available it is not possible to appraise fully the value of penicillin and tyrothricin in relation to otolaryngologic practice, but both of these organic bacteriostatic agents are promising, especially penicillin, because of its solubility, absorbability and extremely low toxicity.

Penicillin.—This chemotherapeutic agent is especially effective against most strains of Staph. aureus and not only against aerobic and anaerobic hemolytic streptococci of group A but also against those of group B (frequently involved in diseases of the ears, nose and throat), as well as against most strains of pneumococci and meningococci, including organisms of these groups which possess a natural or an acquired resistance to the sulfonamide compounds. It is not affected by pus, blood, serum or tissue autolysates, or by the number of bacteria present, although when it is to be injected into the antrums or other closed cavities, the exudates which may dilute the penicillin beyond its bacteriostatic threshold should first be removed.

Since chronic sinusitis is so frequently due to staphylococcic infections, I fully expect that local instillations of penicillin will prove effective in its treatment although frequent applications at close intervals may be required. Recently Robinson and Wallace[9] reported that gauze saturated with a culture of Penicillium notatum may be used in the treatment of localized infections for the production of penicillin in contact with the lesions, and if this proves effective, it may have a special field of usefulness in otolaryngology.

Of special interest and importance to laryngologists, however, are the few reports indicating the effectiveness of penicillin in the treatment of the dreadful fulminating cavernous sinus thrombosis. Undoubtedly penicillin is also the compound of choice in the treatment of cerebral abscess as well as of staphylococcic meningitis. It is also indicated in pneumococcic and streptococcic meningitis, especially if these grave infections are found resistant to sulfonamide compounds. Unfortunately, it appears to be ineffective against meningitis caused by H. influenzae and apparently ineffective against meningitis and other infections due to Friedländer's bacillus.

The administration of penicillin is also indicated in the septicemias due to hemolytic streptococci or staphylococci secondary to phlebitis and thrombosis of the lateral sinus, especially if these septicemias are resistant to treatment with sulfonamide compounds. Undoubtedly it is the most effective chemotherapeutic agent known for the treatment of staphylococcic osteomyelitis of the frontal bones, mastoid processes and other parts.

Intramuscular injection of 15,000 units every three hours may suffice but intravenous injection by the drip method may be required in order to maintain effective concentrations in the blood, because the compound is so rapidly eliminated. Unfortunately, it does not penetrate into the subarachnoid space, so that an intrathecal injection

9. Robinson, G. H., and Wallace, J. E.: An Inoculated Penicillin Dressing, Science **98**:329 (Oct. 8) 1943.

of 10,000 units (1,000 units per cubic centimeter of sterile saline solution) is required every eight to twelve hours in addition to intramuscular or intravenous injections in the treatment of the suppurative meningitides. In this connection I advise that sulfadiazine or some other sulfonamide compound be given orally at the same time in maximum doses, as there does not appear to be any incompatibility and the combination appears indicated in the treatment of these highly fatal infections.

Tyrothricin.—This is an organic and bacteriostatic agent extracted by Dubos [10] from cultures of Bacillus brevis, an aerobic spore-bearing bacterium derived from soil. It is effective against gram-positive organisms, such as some strains of staphylococci, pneumococci, streptococci, diphtheria bacilli and others, including anaerobic streptococci and aerobic streptococci of group B commonly involved in infections of the ears, nose and throat.

Tyrothricin consists of a mixture of at least two substances, namely, gramicidin and tyrocidin. While gramicidin is by far the more active of the two, tyrocidin contributes some bacterial activity to the mixture, and for clinical purposes the separation of the two factors is unnecessary. Tyrothricin appears to exert its effects by inhibiting enzymatic action, retarding growth by lysis of the bacterial cells. The inhibitory effects of blood, serum, pus and tissue autolysates on it are relatively slight; cephalin and related substances found in gram-negative organisms are, however, strongly inhibitory, and hence mixed infections with gram-negative organisms may not respond favorably to this therapy.

Tyrothricin is insoluble in water and is dispensed in concentrated solutions made up in propylene glycol and alcohol which may be diluted to the desired strength by addition of sterile distilled water. For small amounts 1 cc. is diluted with 49 cc. of sterile distilled water, which yields a solution containing 500 micrograms per cubic centimeter. It is without effect when given orally and is both dangerous and ineffective when given intravenously. Consequently, the compound should be only topically applied to the site of infection. Its toxicity for tissues, however, is quite low, and it may be instilled into cavities of the body without untoward effects.

Whether or not tyrothricin will prove of therapeutic value for local instillation or application in the treatment of acute and chronic infections of the sinuses, middle ears and mastoid cells or after intranasal operations and other otolaryngologic procedures cannot be stated at the present time, but it appears worthy of clinical trial. It probably will prove of greatest value when used in cases in which the infection has not penetrated too deeply into the tissues, since, as shown by Bordley and his colleagues [11] no effect takes place unless actual contact is made with the infecting organism.

Patulin.—Patulin, a derivative of Penicillin patulum Bainier, has been described recently by Raistrick and his colleagues.[12] It has been found about equally bacteriostatic for both gram-positive and gram-negative organisms in vitro; it is less active than penicillin against gram-positive bacteria but more active against gram-negative bacteria. It is stated to retain its antibacterial activity in the presence of serum or pus. Solutions of the substance sprayed into the nose seemed to be effective in inhibiting the growth of staphylococci, pneumococci, streptococci, Micrococcus catarrhalis and other pathogens considered to be responsible for the secondary bacterial infections attending the common cold. In a group of 136 treated patients, 57 per cent are stated to have completely recovered within forty-eight hours, compared with 9.4 per cent who recovered in a group of 85 untreated patients. Medical personnel and others working with the substance at the time also used it and noted similar beneficial results. The first experiment was conducted by Gye [12] on himself, with the result that the purulent stage of the common cold, which usually persisted for at least a week, was prevented. The nasal obstruction common in the first stage of the infection was generally relieved, but other symptoms were not affected. No reports are available as yet on the use of patulin in the treatment of acute and chronic sinusitis and other localized infections by topical applications, but it would appear that if these preliminary results are confirmed patulin may prove to be a valuable adjunct to the chemotherapy of diseases of the ears, nose and throat. No ill effects were noted from the local use of the substance in dilutions of 1:5,000, 1:10,000 and

10. Dubos, R. J.: Studies on a Bactericidal Agent Extracted from a Soil Bacillus: I. Preparation of the Agent; Its Activity in Vitro, J. Exper. Med. **70**:1-11 (July) 1939.

11. Bordley, J. E.; Crowe, S. J.; Dolowitz, D. A., and Pickrell, K. L.: The Local Use of the Sulfonamides, Gramicidin (Tyrothricin) and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **51**:936-944 (Dec.) 1942.

12. Raistrick, H.; Birkinshaw, J. H.; Bracken, A.; Gye, W. E.; Hopkins, W. A., and Greenwood, M.: Patulin in the Common Cold: Collaborative Research on a Derivative of Penicillium Patulum Bainier, Lancet **2**:625-635 (Nov. 20) 1943.

1:20,000 although in 1:5,000 dilution it caused painful irritation of the nasal mucosa.

THE CHEMOTHERAPY OF SPIROCHETAL DISEASES

Up to this point I have confined myself to a discussion of the bacterial and viral diseases in relation to otolaryngology, but no discussion could be complete without at least brief reference to the treatment of diseases due to spirochetes, especially syphilis.

It is true that some of the pathogenic yeasts and molds produce diseases of the ears, nose and throat, but chemotherapy has nothing of special value to offer in their treatment except the usual fungicides employed by local application in the treatment of the ringworm and thrush, unless it be sulfanilamide and penicillin in the treatment of actinomycosis, a disease in which these drugs are reported as having yielded encouraging results in some cases. The pathogenic rickettsias do not ordinarily produce diseases in relation to otolaryngology, but even were it otherwise, chemotherapy has nothing to offer against them. It is true, however, that chemotherapy has proved wonderfully effective in the treatment of malaria and other diseases due to animal parasites; fortunately, few of these diseases involve the ears, nose and throat, especially in the United States.

So far as syphilis is concerned, the treatment of the disease in the primary stage characterized by chancres of the nose or the fauces differs in no way from that of primary syphilis elsewhere in the body. Suffice it to state that the diagnosis should be made as promptly as possible, usually with the aid of dark field examinations for Treponema pallidum, so that treatment may be instituted without delay. In this connection it is to be stated, however, that dark field examinations are of little or no value in the diagnosis of chancres of the tonsils or the oral cavity because it is impossible to differentiate between T. pallidum and Treponema microdentium, the latter commonly occurring in the saliva, especially in persons with gingivitis due to spirofusillary micro-organisms. In this connection it is to be emphasized that no otolaryngologist should undertake the treatment of early syphilis unless he is willing to use the continuous method over a period of about fifteen months, employing alternate courses of an organic arsenical and a bismuth preparation, the one by intravenous and the other by intramuscular injection, as recommended by the Clinical Cooperative Group.

In regard to clinically latent and chronic syphilis in general, however, otolaryngologists are properly urged to employ the serologic tests in both private and clinical practice for their detection more commonly than they generally do. Needless to state, a history containing no implication of infection with T. pallidum has little value in excluding its possible presence. Furthermore, respectability is no bar to the penetrability of T. pallidum, as the disease occurs among both the high and the low, irrespective of race, sex or social status. When properly conducted, the approved complement fixation and flocculation tests available at the present time possess a remarkable degree of sensitivity and specificity. Unfortunately, however, falsely positive reactions occur in the best laboratories, although their incidence is largely in relation to the skill and experience of those who conduct the tests since no test can be better than the laboratory conducting it.[13] Undoubtedly there is no one best serologic test for syphilis. Indeed, all serologic surveys conducted by the United States Public Health Service in cooperation with the American Society of Clinical Pathologists have shown that the serologic diagnosis of the disease is best served by using routinely at least two or more tests possessing the maximum of sensitivity consistent with specificity.

Inevitably positive reactions are observed in cases in which the disease is not suspected clinically. Under the conditions they should not be ignored; the tests should be repeated one or more times, since in cases in which the detection of syphilis is not otherwise possible repeated positive reactions may be, and usually are, the only evidence of the disease at a time when prompt institution of proper and adequate treatment may be of inestimable value to the patient. When different laboratories report varying results, it is frequently difficult to reach a decision, but, in my opinion, decision rests with the clinician rather than with the laboratory, since the latter is doing all that can be reasonably expected by conducting the test or tests exactly as described by authoritative serologists and reporting the reactions exactly as observed.

So far as the treatment of chronic latent or tertiary syphilis involving the ear, nose or throat is concerned, it differs in no essential particulars from treatment of the disease elsewhere in the body. Unfortunately, however, there has been no standardization of such treatment as in the case of early syphilis. Indeed, every patient requires indivdual management, and unless the otolaryngologist has acquired special skill and

13. Kolmer, J. A.: The Problem of Falsely Doubtful and Positive Reactions in the Serology of Syphilis, Am. J. Pub. Health **34**:510-525 (May) 1944.

knowledge in the treatment of syphilis of the chronic latent or the tertiary type, it is better to refer the patient to some colleague who has both these qualifications so far as treatment is concerned. Certainly, early syphilis is curable in the great majority of cases when appropriate treatment is given. The old dictum "once syphilitic always syphilitic" is no longer necessarily true. Even chronic syphilis may be arrested and improvement amount to what has been called "clinical cure." It is important to treat the patient rather than his positive serologic reactions.

In Vincent's angina local applications of neoarsphenamine in the form of powder or in 4 per cent solution in 50 per cent glycerin are usually helpful, although in severe infections the compound should be given also intravenously or fuadin intramuscularly. The organic arsenical compounds have also proved highly effective in the treatment of yaws, pinta and the relapsing fevers and sometimes helpful in the treatment of rat bite fever due to infection with Spirillum minus.

2101 Pine Street.

MODERN VIEW OF NEURALGIA REFERABLE TO MECKEL'S GANGLION

REPORT OF CASES SHOWING RELIEF OF PAIN AND SOMETIMES ARREST OF DEVELOPMENT OF ULCERS OF THE CORNEA BY COCAINIZATION OF THE GANGLION

B. R. DYSART, M.D.
PASADENA, CALIF.

In trying to find out the modern point of view with respect to neuralgia due to involvement of Meckel's ganglion, I have read all the articles obtainable written in the last ten years. Twelve articles from the French journals were obtained from the Army Medical Library in Washington by photostatic reproduction.

Much careful work has been done on this fascinating subject. Larsell, Barnes and Fenton [1] reported an experiment in which electrical stimulation of the sphenopalatine ganglion in the rabbit produced vasoconstriction of the vessels in the ear. Photographs were reproduced in which the larger vessels were reduced by 12 to 35 per cent of their size. The small capillaries disappeared entirely. When the cervical sympathetic trunk was cut, vasodilatation occurred, which did not change when the area of the ganglion was restimulated. As a controlling test to see whether fibers were carried through the fifth nerve, the maxillary nerve was cut in other rabbits. No change from the normal response occurred when the area of the ganglion was restimulated. This is favorable evidence for their theory that Sluder's neuralgia is due to vasomotor changes in the area where the pain is felt.

For a long time I have thought that at least in some cases atypical headache and neuralgia were due to vasomotor changes.

About ten years ago I had a patient whose record cannot now be located. She was a dental assistant, about 50 years of age. She would come for relief from a one-sided headache. She mentioned that the affected side of the head, face and arm felt cold. The difference in temperature of the two sides of the face could be easily noted. She never failed to get relief from the headache on cocainization of Meckel's ganglion. The difference in temperature would disappear as the headache disappeared.

There can be no doubt that some of the most severe headaches in the lower half of the head are caused by pathologic disturbances in the sphenoid sinus. I think this type might well be called vidian nerve neuralgia, as recommended by Vail.[2] I can remember 3 patients who had such intense pain just back of the mastoid process that each of them had diagnosed his condition as "mastoid." In each case the pain was relieved by washing pus from the sphenoid sinus.

An interesting case was reported by Gottesfeld and Leavitt.[3] A man of 23 began to have "crocodile tears" in the right eye two years after the development of Bell's palsy of the right side. The tears appeared only at meal time. The duration of the condition before treatment was three years. Cocainization of Meckel's ganglion on the right side would stop the tearing for three or four hours. Injection of alcohol stopped the symptoms for four and one-half months. It was thought that a nerve fiber that originally supplied a salivary gland had been destroyed by the pressure causing Bell's palsy and grew out in a nerve sheath to the lacrimal gland.

The French writers were almost all concerned with the effect of cocainization of the ganglion on ophthalmic symptoms. Alajouanine and Thurel [4] reported:

> The application of Bonain's liquid (equal parts of cocaine, phenol and menthol) over the sphenopalatine ganglion is particularly effective in cases of algesia of ocular origin. Such applications give relief in cases of chronic glaucoma or chorioretinitis and of detachment of the retina accompanied by hypertension of the eye-

From the Department of Otolaryngology of the University of Southern California Medical School.

Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, Jan. 23, 1944.

1. Larsell, O.; Barnes, J. F., and Fenton, R. A.: Relation of Irritation in Region of Paranasal Sinuses to Certain Vasomotor Changes, Arch. Otolaryng. **27**: 266-274 (March) 1938.

2. Vail, H. H.: The Pathways of Reflex Pain in Vidian Nerve Neuralgia, Arch. Otolaryng. **21**:277-284 (March) 1935.

3. Gottesfeld, B. H., and Leavitt, F. H.: "Crocodile Tears" Treated by Injection into the Sphenopalatine Ganglion, Arch. Neurol. & Psychiat. **47**:314-315 (Feb.) 1942.

4. Alajouanine, T., and Thurel, R.: La sympathicothérapie locale (ganglion sphéno-palatin), Rev. d'oto-neuro-opht. **13**:645-664 (Nov.) 1935.

ball. Furthermore, they seem to have a favorable influence on hypertension of the eyeball. . . . The application of Bonain's liquid rapidly establishes control of crises characterized by the flow of tears and also stimulates the process of recovery in tardy cases of conjunctivitis and neuroparalytic keratitis of sympathetic origin.

They advised that the medicament be left in place over the ganglion for thirty minutes to one hour. They claimed that actual necrosis of the ganglion fibers occurs.

Cerise and Thurel [5] reported good results in cases of ulcers of the cornea and in 3 cases of retrobulbar neuritis when the preparation described was applied over Meckel's ganglion.

Luedde,[6] professor of ophthalmology at the St. Louis University School of Medicine, reported several cases of glaucoma in which definite lowering of intraocular tension was obtained by cocainization of the sphenopalatine ganglion or injections of 2 per cent procaine hydrochloride into the region of the ganglion. He did not recommend this treatment as a substitute for surgical reduction of tension in glaucoma but stated that by this means a period of relief may be had in which to investigate possible causes of the glaucoma and decide on a favorable time for operation. He reported a case of ciliary spasm in a teacher of 21. During the spasm the eyes were myopic to the extent of 5 diopters. Cocainization of the ganglion by Dr. Sluder brought the eye to a normal state, in which it remained for six years. The first attack lasted six months before treatment. Another attack, six years later, was treated by Dr. A. W. Proetz, and again sphenoiditis was found. Again cocainization of Meckel's ganglion gave the greatest relief. A third attack, nine years later, was relieved by packs applied to the ethmosphenoid region.

I should like to report here one of the most interesting cases I have observed. For the past eight years I have been observing a woman with recurring ulcers of the cornea. During the past twenty years she has had probably more than one thousand corneal ulcers.

CASE 1

On July 24, 1935, this patient, 47 years old, who was employed in a laboratory of pathology, was waiting to see her ophthalmologist, Dr. W. H. Roberts, my former associate. She mentioned that in addition to the pain of a beginning ulcer she had a severe occipital headache. In hopes of relieving the headache, I cocainized the sphenopalatine ganglion. To the intense astonishment of all of us, redness, pain, photophobia and lacrimation disappeared completely within three or four minutes. The patient said that it was as if a hot coal of fire had been taken out of her eye. This relief was complete for about five hours. The symptoms then returned, and another treatment was given, with no return of symptoms.

The ulcers had first started in 1923 from no known cause. The patient had undergone partial thyroidectomy in 1914. For a while she had taken thyroid extract but at the time of my examination had a normal basal metabolic rate. She had had mild pulmonary tuberculosis in early adult life, but frequent roentgen examination of the chest showed no later activity. She was highly allergic to tuberculin, and after a tuberculin test had been done in the laboratory, she noticed puffing of her eyes. Otherwise there was no definite history of allergy. In 1926 she had bilateral maxillary sinusitis requiring a few irrigations, but in roentgen study and careful observation later further infection could not be detected.

In the next attack a few weeks later the patient was not treated until six hours after the attack began. A corneal ulcer was visible, and a staining defect was demonstrated by application of fluorescein. Two cocainizations the first day and one the second day cleared up this ulcer. Previously, with the usual ophthalmic treatments the ulcers would always persist for five to ten days. Thirteen ulcers had been so severe that Dr. Roberts had treated them with the electric cautery. There was slight scarring visible in two or three spots on the cornea where cauterization had been done. It was soon observed that if the ulcer was treated twelve or more hours after onset it would take three or four days to heal.

During the eight and one-half years that have followed, many attempts have been made to find the cause of these ulcers. Several internists have carefully examined her. Dr. Edward Rosenow Sr., of the Mayo Clinic, prepared an autogenous vaccine from a blood culture which showed a growth of Streptococcus viridans. Even the weakest dilutions produced general aching and local swelling. Another vaccine was made from a culture of material from the throat which showed Str. viridans. The same severe reactions occurred with no apparent benefit.

Dr. L. L. Henry, an ophthalmologist, decided that her disease was mild latent pulmonary tuberculosis, and at his request Dr. Francis Pottenger Sr. tried desensitizing the patient with minute doses of tuberculin. Rather severe reactions followed the smallest dose. Dr. Henry then had the patient remain in bed six months in hopes of reducing the possible latent tuberculosis. While she was in bed there were no ulcers; as soon as she was up and at work again, the ulcers returned. Fatigue was evidently a factor.

In 1941 the patient went to Seattle by automobile. On the return trip an attack started in a small town where treatment was not available. She found she could easily stop the attack by cocainizing the ganglion herself. I had coached her in the technic and had given her an applicator and a small supply of cocaine. Had the patient not been of a stable disposition, this possibility of starting a habit would not have been taken. Since then she usually has stopped the attacks herself.

In 1942 Dr. Hunter Sheldon, after careful examination, suggested injections of histamine phosphate. No improvement was noticed.

The patient has had fewer attacks in the last year, possibly because her general condition is better. Cocainization still stops beginning attacks, but slightly longer applications are required. The symptoms from the ulcers

5. Cerise, L., and Thurel, R.: La phénolisation du ganglion sphéno-palatin dans les névrites rétroubulbaires, Rev. d'oto-neuro-opht. **15**:65-70 (Jan.) 1937.

6. Luedde, W. H.: Relation of Meckel's Ganglion to Accommodation and Intraocular Tension, Illinois M. J. **79**:199-209 (March) 1941.

disappeared at first in three or four minutes. Now they disappear in eight to ten minutes.

Injections of alcohol or surgical removal of the ganglion have been suggested, but the patient prefers to avoid these measures. She states that the relief of pain from cocainization has been nothing short of life saving.

The explanation of the healing power of cocaine in case 1 may lie in relief of vasomotor spasm of capillaries supplying the cornea. I have observed 2 other cases of prolonged ulceration of the cornea in which pain was relieved by cocainization of the ganglion, but there was no definite influence on the healing of the lesion.

CASE 2

On Oct. 19, 1939, a woman of 35 was referred to me with the request a search be made for a possible focus of infection. She had small multiple ulcers on both corneas, possibly herpetic. The pain was so intense that she had had little sleep for two days in spite of the use of narcotic drugs. Search for a focus of infection revealed none, but cocainization of both ganglions gave complete relief of pain within five minutes. She reported the next day that the relief had lasted through the night. There was no apparent improvement in the appearance of the ulcers. The ulcers persisted several weeks in spite of cocainization of the ganglions five more times and treatment of the eyes. The cocainization never failed to give considerable relief of pain.

CASE 3

A man of 77 was referred Oct. 19, 1939, with the request that a search be made for a possible focus of infection in the sinuses. He had been suffering for fourteen days with an ulcer in the right eye. The pain was moderately severe. Complete relief of pain was obtained five minutes after cocainization of the right ganglion. No focus of infection could be found. A second treatment was given the next day with relief of pain. No improvement was noticed in the appearance of the ulcer, and since the pain was not great, he was not sent back for further treatment.

I think that it is possible that earlier or more frequently repeated cocainization might have produced better results in cases 2 and 3.

The following case is one of pain back of the eye with no ulceration or lesion of the eye.

CASE 4

A physician, A. L. R., about 35 years old, came to me on Aug. 25, 1938. He had been suffering from intense pain back of the left eye at frequent intervals for four or five months. Dr. R. M. Decker had given him relief on several occasions by cocainizing the sphenopalatine ganglion. Relief usually lasted three to four hours. He had been able to get partial relief by a hypodermic injection of codeine phosphate, 1 grain (0.06 Gm.). For two weeks he had been having almost continuous attacks and was getting desperate. He requested injections of alcohol or anything to get relief. He said he felt as though some one were driving a large nail right through his eye from behind. The usual search for foci of infection led to four infected teeth. These were removed, and except for a slight attack six days later, requiring cocainization, he was free of pain for one year. A year later he had another attack. Instead of coming to me, he had another tooth removed concerning which a roentgenogram had aroused suspicion. His pain was relieved, and he had no more during the following four years.

TECHNIC

Just a word about technic. When a patient is being treated for the first time and his tolerance to cocaine is not known, one should proceed with care. I usually spray a little 2 per cent cocaine hydrochloride into the middle meatus and wait a few minutes to see whether there is any elevation of the pulse rate. I then apply 1 drop of 10 per cent cocaine hydrochloride by means of a fine flexible copper applicator. This is left in place five minutes.

Applicators may be easily made of fine copper wire. If copper wire is not available, fine wire of any material may be used if it is not too stiff. The wire is cut into lengths 5 inches (12.5 cm.) long. A small loop about ½ inch (1.27 cm.) in diameter is made at one end for holding. The tip is then roughened by crushing it in a strong hemostat with corrugated jaws. A small wisp of cotton large enough to absorb only a small drop of the cocaine preparation is used. The tip is then bent into a curve about the same as that used on a eustachian catheter. The turbinates should be shrunk thoroughly with a vasoconstrictor drug before an attempt is made to reach the ganglion. I prefer one or two puffs of 2 per cent cocaine hydrochloride directed toward the middle turbinate. Then, with the tip turned up, the applicator is slid along the lower edge of the middle turbinate without touching it or the septum if possible. As the back wall of the nose is reached, the tip is rotated outward and lifted upward to lie just back of the attachment of the middle turbinate. The patient with the frequently recurring ulcers noticed that when the applicator was placed too high a slight pain was felt in the top of the head and the relief of the symptoms due to the ulcer was only partial. Another patient gets relief only if the application is made slightly higher than the usual spot. A third patient, a retired pathologist, notices a severe burning sensation when the proper spot is reached. He is the only patient who has mentioned this. If there is no improvement, a saturated solution of cocaine hydrochloride (90 per cent) is used and is applied for five minutes. At any rise of pulse rate or a complaint of feeling a little faint, the treatment is stopped. If the patient seems to be definitely reacting to cocaine, I give 3 or 4 teaspoons of elixir of phenobarbital U. S. P. For severe reactions one should have on hand barbital sodium or sodium pentothal for intravenous injection. The barbiturates are antidotes for cocaine poisoning. Reactions are rare. If a reaction occurs and it is important to continue the investigation, I have the patient take ¾ to 1½ grains (0.048 to 0.09 Gm.) of pentobarbital sodium thirty minutes before the next attempt. This is the technic (slightly modified) described by Dr. George Dock, who was treated by Dr. Sluder for migraine headaches.

I have one patient whose travels have taken him to most of the cities of the United States. He is subject to migraine headaches but is able to get immediate and lasting relief from cocainization of Meckel's ganglion if this is done properly. He says that "the average specialist takes an applicator about as big as a poker and manipulates it as if he were stirring a fire." The pain produced is great and the relief is slight. Incidentally, he recently spent two weeks in Johns Hopkins Hospital, and after a thorough study, was given histamine with good results. As long as he takes a little histamine phosphate every two weeks, he has no headache. His symptoms are, to me, not typical of the

vasomotor type described by Horton,[7] but are typical of migraine headaches.

In closing let me again quote Luedde [6]:

The explanation for the relief obtained in ciliary spasm is not found in the actual function of the sphenopalatine ganglion, but apparently depends on the accessory sensory fibers which pass through it without being a part of its true ganglionic structure. These sensory fibers may be effectively blocked by the direct application of cocaine or the injection of novocaine into the region of the ganglion. . . .

Meckel's ganglion then is in reality an innocent bystander, but being at the cross roads where both sympathetic fibers and sensory fibers are concentrated, it has been looked upon as the arch offender by Sluder, Ewing and others. Our lesson in this connection would appear to be that ophthalmologists can no longer successfully remain isolationists. The cause of glaucoma is to be sought out by considering anything that may modify capillary osmosis within the eye ball.

Does all glaucoma originate in the nose? No. Can nasal disorders have an influence in the onset or course of the intraocular hypertension? Emphatically, yes.

By the same token, rhinologists must cooperate with ophthalmologists, doing their part in the control of these conditions.

CONCLUSIONS

1. The pain of ulcer of the cornea and in many cases atypical headache and neuralgia may be relieved by cocainization of Meckel's ganglion.

2. In some cases the progress of corneal ulcers or other lesions is favorably influenced.

3. The technic outlined gives the relief described.

4. Cocainization of Meckel's ganglion probably achieves its effects by intercepting nerve impulses and not by influencing any pathologic process in the ganglion.

65 North Madison Avenue.

7. Horton, B. T.; MacLean, A. R., and Craig, W. M.: New Syndrome of Vascular Headache: Results of Treatment with Histamine, Proc. Staff Meet., Mayo Clin. **14**:257-260 (April 26) 1939.

THROMBOSIS OF THE CAVERNOUS SINUS WITH HEMOLYTIC STREPTOCOCCIC BACTEREMIA

TREATMENT BY INTRAVENOUS INJECTION OF SULFADIAZINE AND PENICILLIN, WITH RECOVERY

JONAS W. WOLF, M.D.

YONKERS, N. Y.

Prior to the era of chemotherapy, true cases of thrombosis of the cavernous sinuses were invariably fatal. None that I know was ever reported with cure. Medical treatment was of no avail. Surgical procedures, such as ligation of the facial vein, met with similar results. Since the introduction of sulfonamide compounds, however, a number of cases of thrombosis of the cavernous sinus have been reported with cure.[1]

Before presenting my case I shall review briefly the etiology and trace the course of the thrombus from the source of infection to the cavernous sinus. Infection originates from anterior, intranasal, posterior and inferior foci. In this case the port of entrance was anterior: a furuncle on the bridge of the nose. The septic thrombus spread to the angular, to the supraorbital, to the supratrochlear and to the ophthalmic vein, emptying into the cavernous sinus. There it extended to the opposite side, affecting the other eye.

REPORT OF A CASE

W. P., a boy 10 years of age, had no history of illness except that at the age of 4 he had suffered a severe extensive burn of both thighs.

On Nov. 1, 1943 I was summoned to his home, where I obtained the following history from his mother. For the past four weeks the child had been suffering from multiple furuncles on both legs. He had not been seen by a physician but was receiving home treatment. On October 29 his mother noticed a slight redness on the bridge of the nose. The following day a furuncle appeared on the left side of the bridge of the nose and the left eye became red. October 31 the boy had a chill and felt feverish, and the upper eyelid on the left side became swollen. He found difficulty in opening the eye. That same night he became very ill, with high temperature, chills and delirium; the left eye was completely closed, and the eyelid was red.

On my arrival I found a male child 10 years of age, seemingly very ill, with a temperature of 104.2 F., taken rectally, and a pulse rate of 130. There was cellulitis of the left upper eyelids; the eyeball was stationary and hard; the pupil did not react to light. The bridge of the nose was swollen and showed a necrotic area about 5 mm. in diameter. Otherwise physical examination gave negative results. The child was immediately hospitalized.

The physical findings at the hospital were essentially the same except that in the afternoon of the same day the right eyelid became edematous and toward evening became completely closed. The laboratory findings on admission were as follows: The reaction of the urine was acid. The urine showed a specific gravity of 1.028, albumin (3 plus), acetone (1 plus) and no sugar. There was an occasional granular cast. There were 6 to 10 pus cells per high power field and many blood cells. Examination of the blood showed a hemoglobin content 82 per cent of normal, 4,530,000 erythrocytes per cubic millimeter, a color index of 0.91 and 22,300 leukocytes.

Course, Detailed Treatment and Progress of Case.—Immediately on admission, November 1, the child received 5 Gm. of sodium sulfadiazine intravenously and 3 Gm. of sulfadiazine by mouth in divided doses. The temperature continued to fluctuate from 103.1 to 104.5 F; the pulse rate was about 120 and the respiratory rate 40 per minute. The child was delirious and unruly.

On November 2 his urine remained unchanged except for albumin (1 plus), a specific gravity of 1.018 and some sulfadiazine crystals. The hemoglobin content was 65 per cent, the erythrocyte count 3,520,000, the color index 0.92 and the white cell count 19,200. Culture of material from nose and throat showed a growth of the hemolytic streptococcus. There was cellulitis of both upper eyelids and of the forehead, extending into the scalp; both eyeballs were fixed, with no reaction of the pupils. There were suggestive rigidity of the neck, absence of knee jerk, and suggestive ankle clonus. The temperature ranged from 103.1 to 105.8 F.; the pulse rate was 120 and the respiratory rate 30 to 50. The same morning the patient received 2.5 Gm. of sodium sulfadiazine intravenously. During the afternoon a half million units of penicillin was obtained, and the initial dose of 60,000 units was given intravenously by the continuous drip method. The administration of sodium sulfadiazine was discontinued.

November 3 the child showed a great deal of cerebral irritation; he was delirious and constantly talking. The cellulitis of the eyelids and the forehead increased in severity and extended farther into the scalp. Both eyeballs were very hard. The rigidity of the neck was about the same. The temperature ranged from 103.8 to 104.9 F.; the pulse rate was 120 to 130 and the respiratory rate was 40 to 50. A blood culture showed a growth of numerous colonies of the hemolytic streptococcus. Sixty thousand units of penicillin was given over a period of twenty-four hours by continuous drip.

November 4 the patient's condition was not improved. The temperature remained constant at about 104 to 104.8 F. The child was uncooperative and nonresponsive.

1. Seydell, E. M.: Recovery from Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **30**:429 (Sept.) 1939. Morrison, L. F., and Schindler, M.: Cavernous Sinus Thrombosis, ibid. **31**:948 (June) 1940. Skemp, H. L.; Afremow, M. L., and Rhoads, P. S.: Cavernous Sinus Thrombosis, ibid. **34**:1025 (Nov.) 1941.

The neck was more rigid. The cellulitis of the eyelids and the forehead remained the same. There also began to appear marked concentration of fluids in tissues, and the patient began to complain of pain in the chest. Examination of the lungs revealed some scattered rales. A report on the urine gave the specific gravity as 1.010 and the albumin as 3 plus and mentioned hyaline casts.

As this time I began to realize that my associates and I were dealing with a malignant bacteremia, and I felt that I had no right to confine myself to one drug. Sodium sulfadiazine was reinstituted so that on the same day the patient received 60,000 units of penicillin and 5 Gm. of sodium sulfadiazine intravenously.

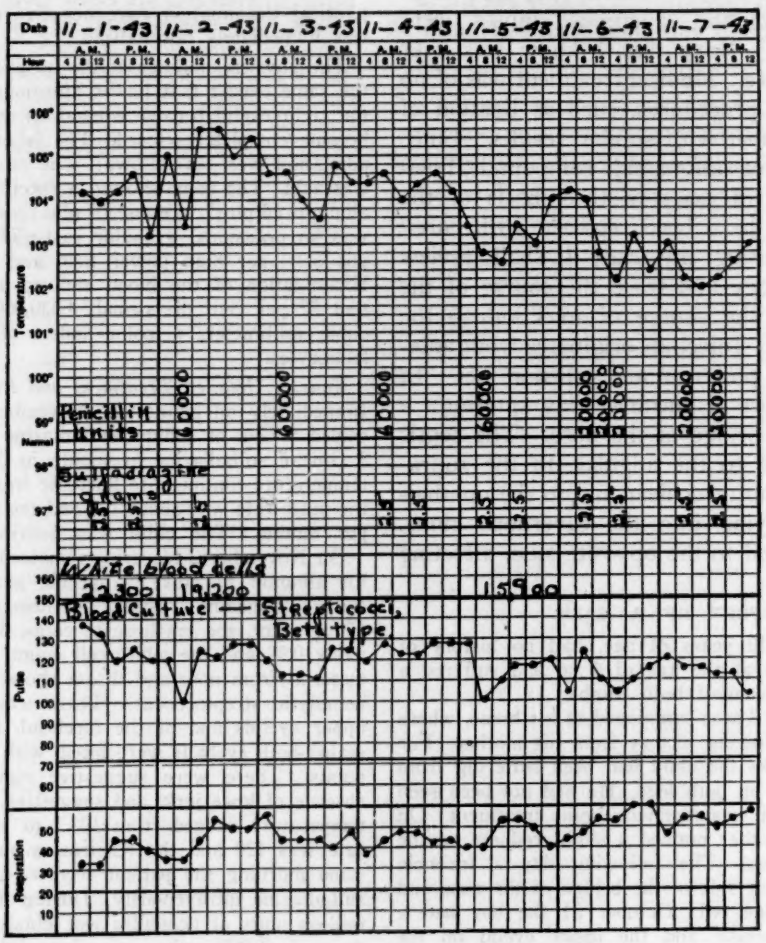


Chart 1.—Clinical observations at height of bacteremia, with administration of both penicillin and sodium sulfadiazine.

The following day, November 5, he showed slight improvement. The temperature ranged from 102.3 to 104 F., the pulse rate from 100 to 120 and the respiratory rate from 40 to 50. The boy was less irritable. The blood sulfadiazine concentration was 18 mg. per hundred cubic centimeters. The hemoglobin content was 64 per cent, the red cell count 3,710,000 and the leukocyte count 15,900. The patient began to have some edematous rales in his lungs, and consequently the continuous drip method was discontinued. Divided doses of sodium sulfadiazine and penicillin were given intravenously, more concentrated solutions being used.

November 6, the child continued to improve but was still somewhat irritable. The rigidity of the neck was much less; the ankle clonus was absent; the cellulitis of the right eye was beginning to pale, and the temperature ranged from 102 to 104 F. Sixty thousand units of penicillin and 5 Gm. of sodium sulfadiazine were given intravenously in three divided doses. The urine of that day showed a specific gravity of 1.020, a trace of albumin, an occasional red blood cell and a moderate number of sulfadiazine crystals. The output of urine was good.

November 7 a definite decline in temperature began to take place, according to the chart, the curve ranging from 102 to 103; the pulse rate was 110; the respiratory rate was still high, 50 per minute. The cellulitis definitely began to subside. The right eyelid was much softer, and the left eyelid began to pale. A blood culture was negative. The hemoglobin content was 65 per cent, the red cell count was 3,600,000 and the leukocyte count was 17,300. The urine was about the same except that there were no sulfadiazine crystals. Treatment consisted of 5 Gm. of sodium sulfadiazine and 40,000 units of penicillin, given in two divided doses.

November 8 the temperature fluctuated between 101 and 102.8 F. The child was somewhat irritable but more cooperative. He continued to complain of pain in the chest. The swelling of the right eye was gradually subsiding; the eyeball was moving to and fro, and he was able to recognize objects. The urine showed a specific gravity of 1.014, albumin (1 plus), acetone (1 plus), occasional granular and hyaline casts and 10 to 15 pus cells. Five grams of sodium sulfadiazine

and 60,000 units of penicillin were given in divided doses.

November 9 the patient's mentality was clearer. He continuously complained of pain in the chest. A roentgen examination showed the heart slightly enlarged and revealed areas of consolidation in the costophrenic angle of the right lung, which the radiologist interpreted as pulmonary thrombi. The left lung showed moderate bronchopneumonia. The temperature ranged from 101 to 103.2 F. The pulse and respiratory rates remained at about the same level as on previous days. The cellulitis of the forehead and the left eye showed definite improvement. The right eye was able to open and the patient noticed objects and surroundings. Sedation was given for pain, and sodium sulfadiazine, 5 Gm., and penicillin, 20,000 units, were given intravenously. Also intravenously, and sulfadiazine, 6 Gm., was given by mouth in divided doses.

November 11 the child continued to improve. The temperature ranged from 100.8 to 102.3 F. The respiratory rate was 40, and the pulse rate ranged from 100 to 110. The blood culture was negative. The urine was the same as on the previous day. Twenty thousand units of penicillin was given intravenously, and 3 Gm. of sulfadiazine was given by mouth in divided doses.

November 12 the right eye was completely open; the left eye, partially open. The edema of the forehead had completely subsided. The mentality was clear. The temperature was 100.2 to 102.2 F. The hemoglobin content was 53 per cent, the red cell count was 3,640,000 and the leukocyte count was 18,200. The urine was the

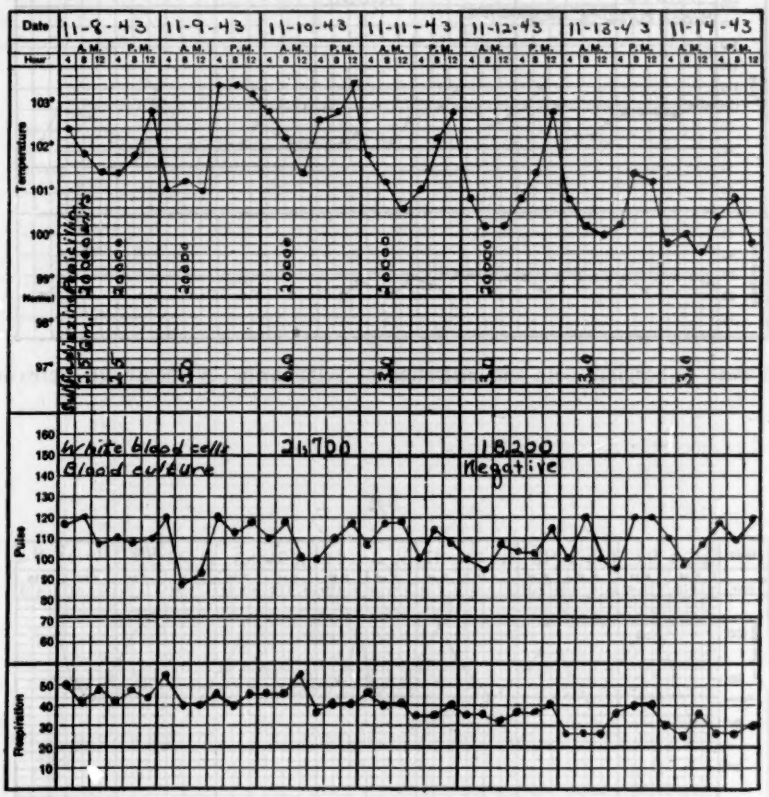


Chart 2.—Clinical observations in second week of the stay in the hospital showing beginning subsidence of disease.

1 Gm. of sulfadiazine was given by mouth every four hours, so that in twenty-four hours the patient received a total of 8 Gm. of sodium sulfadiazine. The urine remained about the same, with no evidence of any crystals.

November 10 the child's mentality was much clearer. The right eye was completely open, with good vision. The patient was able to open his left eye; the left eyeball was movable, and there was some sight. The child was elated over the fact that he could see. The hemoglobin content was 54 per cent, the red cell count was 3,480,000 and the leukocyte count was 21,700. This count was most likely explained by the bronchopneumonia. The blood concentration of sulfadiazine was 8.5 mg. per hundred cubic centimeters. The urine was the same as on the previous day except for a few sulfadiazine crystals. Penicillin, 20,000 units, was given same as on the previous day except for an occasional granular cast. The treatment was the same as on the previous day. Up to this day a total of 500,000 units of penicillin had been given.

November 13 the child continued to progress satisfactorily. The mentality was clear. All neurologic reactions were normal. The right eye recovered completely. With the left eye the patient saw well, but there was still some paralysis of the third, fourth and sixth nerves, so that he could not completely raise the upper eyelid and showed some strabismus. The temperature was 100 to 100.2 F., the pulse rate remained rapid and the respiratory rate ranged between 30 and 40. The urine was the same as on the previous day except for some sulfadiazine crystals. The blood sulfadiazine concentration was 5.1 mg. per hundred cubic centimeters. Sulfadiazine, 3 Gm. in divided doses, was given by mouth.

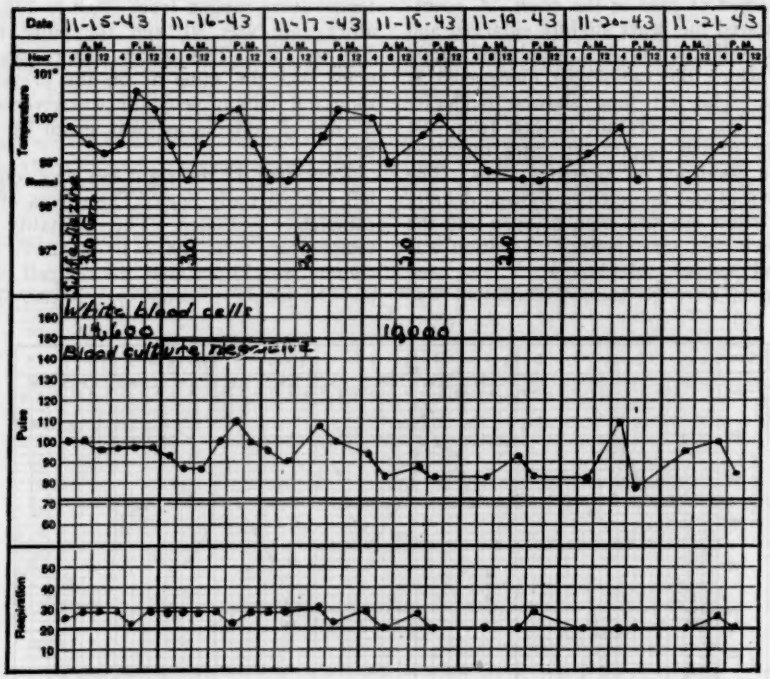


Chart 3.—Third week in hospital, with administration of sulfadiazine by mouth in decreasing amounts.

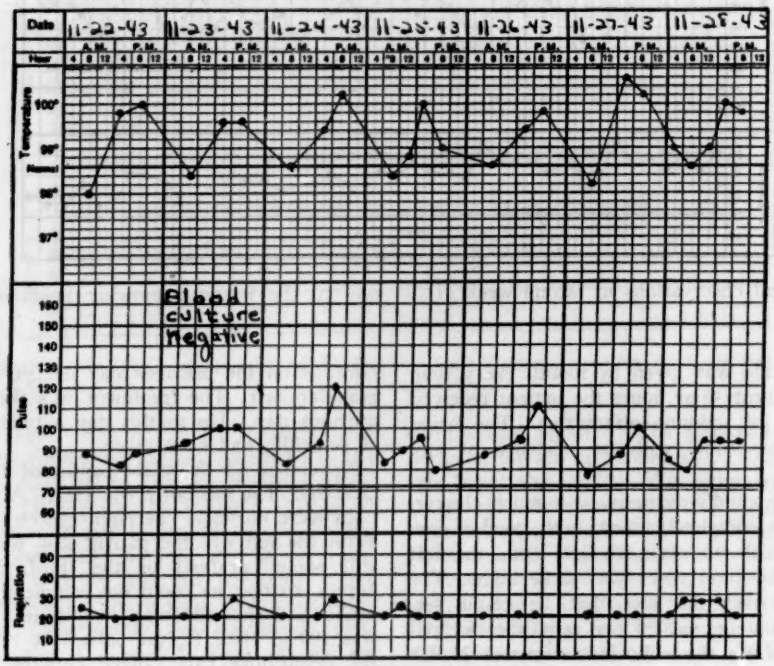


Chart 4.—Fourth week.

November 14 to November 18 the child continued to do well. The temperature ranged between 98.6 and 100.2 F. The treatment remained the same, 3 Gm. of sulfadiazine by mouth.

November 15 the hemoglobin content was 64 per cent, the red cell count was 3,360,000 and the leukocyte count was 14,600.

November 18 the hemoglobin content was 70 per cent, the red cell count was 3,450,000 and the leukocyte count was 10,000. The administration of sulfadiazine was gradually decreased until on November 19 it was entirely discontinued.

November 28 the temperature, the respiratory rate and the pulse rate remained in the normal range. The blood culture was sterile. The hemoglobin content was 78 per cent, the red cell count was 3,820,000 and the leukocyte count was 11,300. The urine was normal with the exception of 4 to 5 pus cells. The edema of both eyes had completely subsided except for that of the left upper eyelid; a slight discoloration and a partial weakness of the upper lid were still noticeable. The child was up and around, with normal vision and clear mentality.

December 3 the hemoglobin content was 80 per cent, the red cell count was 4,230,000 and the white cell count was 9,900; there was slight anisocytosis; a differential count showed 2 per cent stab forms, 1 per cent eosinophils, 41 per cent segmented forms and 56 per cent lymphocytes. The following day the child was discharged as cured. A follow-up visit ten days later showed that he had completely recovered from the paralysis of the oculomotor nerve and had complete control of the levator palpebrae superioris muscles.

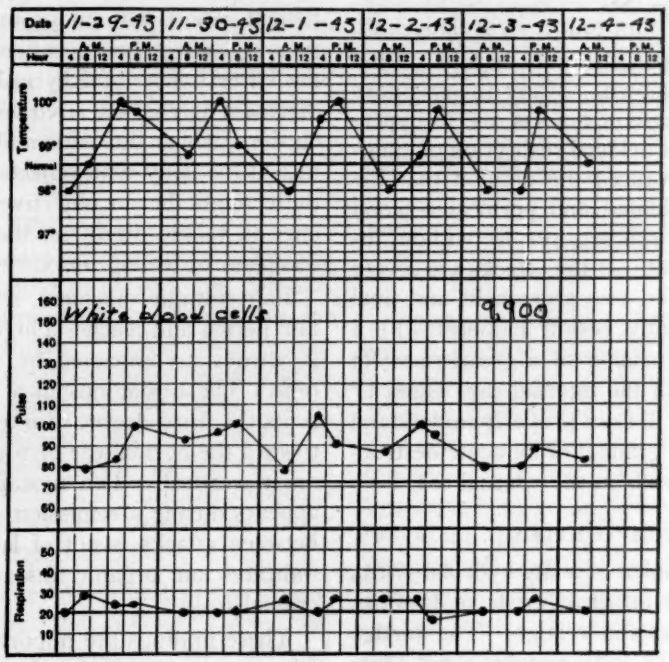


Chart 5.—Fifth week, with discharge of patient as cured.

SUMMARY

A boy of 10 years made a complete recovery from thrombosis of the cavernous sinus with multiple thrombi in the right lung and kidney and hemolytic streptococcus bacteremia. The source of infection was a furuncle on the bridge of the nose. The therapeutic agents were sulfadiazine and penicillin. A total of 500,000 oxford units of penicillin and 64 Gm. of sulfadiazine was administered in a period of nineteen days. No ill effects were noted during the course of treatment. Apologies are hereby in order for not confining myself to one therapeutic agent. However, considering the severity of the conditions faced and the failure of sulfadiazine therapy in a previous case in the same hospital under another physician's care, I felt that the risk was too great for me to gamble with life by depending on one drug. Furthermore I believe, although I may be grossly criticized for the following statement, that sulfadiazine given in large doses to produce a blood level of 18 mg. or more per hundred cubic centimeters had a definite curative effect on the bacteremia in this case. The penicillin, on the other hand, had a definite effect on the septicemia, caused especially by staphylococci, which were undoubtedly present in the original focus. I also wish to point out that not until the blood level was 18 mg. plus did the disease show signs of abating and that when either drug was used exclusively no change in the course of the disease was noted. Thus it is apparent that by the use of large doses of a sulfonamide drug in combination with penicillin, plus diligent and careful observation, thrombosis of the cavernous sinus, a disease looked on in the past as inevitably fatal, may now be cured.

49 Park Avenue.

PRESENT STATUS OF DIAGNOSIS AND MANAGEMENT OF MÉNIÈRE'S SYNDROME

HANS BRUNNER, M.D.
CHICAGO

PATHOLOGY

Ménière's syndrome, according to recent studies, is due to an acute increase of fluid within the internal ear, particularly within the endolymphatic canal of the inferior part of the internal ear.[1] These changes will be called "hydrolabyrinth" to emphasize the similarity to hydrocephalus. As in hydrocephalus, probably more than one mechanism can produce hydrolabyrinth.

The source and the chemical nature of the excess fluid is not known. Many investigators believe that the increase of fluid is due to hypersecretion and perhaps delayed resorption of endolymph. I [2] have advanced the opinion that the fluid escapes from dilated and permeable blood vessels. Hydrolabyrinth may resolve without injury to the internal ear. If the attacks are severe or prolonged, however, two permanent and final changes will eventually result, namely: (1) a fixed dilatation of a smaller or a greater part of the inferior portion of the internal ear, which I [3] described although I did not properly understand it in 1922, and (2) a gradual or an acute destruction of the sensorial cells in the internal ear.

SYMPTOM COMPLEX

The symptom complex of acute hydrolabyrinth causing the Ménière attack consists of tinnitus, diminution of hearing and vertigo. The vertigo is without exception of a labyrinthine type. Only vertigo of the type which can be produced by the usual clinical methods of examining the labyrinth should be considered as labyrinthine. From this point of view I [4] have divided the labyrinthine vertigo into (*a*) the whirling type and (*b*) the tactile type. Whirling dizziness is characterized by a sensation of turning which originates in the patient's eyes when the visible objects are turning or in the patient's musculature when the turning sensation is felt in the patient's body. It is not necessary to search for the direction of the rotatory motion, but it is necessary that the patient report without prompting a sensation of turning and not a sensation of oscillation since the latter is not of labyrinthine origin. Tactile dizziness is characterized by the incorrect conclusions which the patient draws from the contact of his body with objects in space. Although there might be no objective disturbance of balance in tactile dizziness, the patients frequently complain of feeling intoxicated.

Labyrinthine dizziness, which lasts for a certain period and reaches a high degree of intensity, is always accompanied by spontaneous nystagmus. The nystagmus in a Ménière attack presents the third degree of intensity and is directed toward the normal ear or toward the affected ear or is vertical. The nystagmus gradually disappears during a remission. Marked nystagmus between attacks is not of labyrinthine origin; it indicates an organic disease in the posterior cranial fossa.

These findings are important for two reasons: First, patients suffering from hysteria may complain of severe dizziness. In instances of hysteria there is never spontaneous nystagmus. Second, a Ménière attack frequently produces a state of anxiety. Some neurologists speak of a Ménière neurosis. In instances of this, too, there is never spontaneous nystagmus nor can it be elicited by quick movements of the head.

In a typical Ménière attack there are symptoms involving both the cochlea and the labyrinth. Deafness and tinnitus usually increase during the attack of vertigo. In rare instances the patient reports the contrary observation. Just as rarely the patient notices deafness and tinnitus for the first time during the first attack of vertigo. Under all circumstances the typical Ménière attack presents labyrinthine as well as cochlear symptoms.

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine.

Read at the meeting of the American Laryngological, Rhinological and Otological Soc., Middle Section, Jan. 12, 1944.

1. The inferior part of the internal ear includes the cochlea and the sacculus.

2. Brunner, H.: Ménière's Syndrome, Laryngoscope **49**:877, 1939.

3. Brunner, H.: Ueber einen Fall von Paget'scher Krankheit, Monatschr. f. Ohrenh. **56**:810, 1922.

4. Brunner, H.: Allgemeine Symptomatologie des N. Vestibularis, in Alexander, G.; Marburg, O., and Brunner, H.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 1, p. 939.

The evaluation of symptoms becomes difficult when the patients complain only of vertigo or only of deafness and tinnitus. In both instances the attacks might be typical Ménière attacks if in a later period the attacks of vertigo are joined by cochlear symptoms and vice versa. The time which elapses between the first attack of vertigo and the first appearance of cochlear symptoms may be several years. Attacks of vertigo without cochlear symptoms may be due to a cerebellar tumor; attacks of tinnitus and deafness without vertigo, to otosclerosis. In these instances the differential diagnosis is often difficult. Errors of treatment are easily made since the treatment of simple labyrinthine or cochlear attacks differs from that of Ménière attacks.

During a remission the patient may be entirely normal. In cases of progressive disease, however, slight tinnitus and vertigo may persist. Clinically one notes a diminution of hearing of different degree and different type and a labyrinthine hypoexcitability, which frequently is preceded by labyrinthine hyperexcitability.

ETIOLOGY

The cause of Ménière attacks and of Ménière's syndrome may be found to be systemic, cerebral or aural. The syndrome is then called "symptomatic Ménière's syndrome." When no cause can be determined, the condition is designated as "idiopathic Ménière's syndrome." Cases of idiopathic Ménière's syndrome are in the minority, but they have stimulated great interest concerning their etiologic factor. At present two concepts are under discussion: (*a*) the metabolic theory and (*b*) the vascular theory.

The Metabolic Theory.—Furstenberg, Lashmet and Lathrop found that Ménière's syndrome is due to retention of sodium. They advised reduction of the intake of sodium. The patient is placed on a sodium-free diet, and an acid-forming salt, ammonium chloride, is administered to insure the maximum elimination of sodium. Mygind and Dederding,[5] on the other hand, have expressed the belief that the essential factor in Ménière's syndrome is that water is retained, generally as well as locally, but above all in the ears. They advised restriction of the intake of fluids. The metabolic concept is not supported by microscopic observation of the ear nor, as emphasized by Furstenberg, Richardson and Lathrop,[6] by chemical examination of the blood. Furthermore, it is difficult to understand that a disturbance of the general metabolism can produce trouble limited to the ear. The practical results obtained by the types of treatment described are not convincing. Mygind and Dederding[5] reported a definite cure in 42.7 per cent of cases and a transitory cure in an additional 52.8 per cent of cases. The results obtained by Furstenberg and associates[6] are presented in the following tabulation:

Total Number of Cases	Cases in Which Condition Was Cured or Improved	Cases in Which Condition Was Unaltered
95	84%	16%

To appreciate these figures properly I present the figures of von Frankl-Hochwart,[7] published in 1905, to which a few of my cases are added. In all these cases the treatment consisted of inflation of air into the tympanic cavity, rest, climatic-hygienic measures, hydrotherapy, galvanization of the skull and administration of small doses of bromides.

Total Number of Cases	Cases in Which Condition Was Cured or Improved	Cases in Which Condition Was Unaltered
60	81.6%	18.4%

Among the 60 cases just cited there were 36 with a chronic adhesive process of the middle ear and thus these were instances of symptomatic Ménière's syndrome. There may have been other instances of symptomatic Ménière's syndrome in this series. In the statistics of Furstenberg and associates[6] cases of idiopathic Ménière's syndrome are apparently included. However, though symptomatic and idiopathic Ménière's syndrome differ from the standpoint of etiology, they probably do not from that of pathology. The average period during which the patients were observed was about thirty-four months for the cases of Furstenberg and associates[6] and twenty-seven months for the cases of von Frankl-Hochwart.[7]

It is difficult to determine whether or not the cures and improvements reported by these authors were actually ascribable to treatment. Not infrequently the attacks of vertigo cease spontaneously. In other instances attacks cease after destruction of one or both labyrinths has occurred. If a proper evaluation of the treatment is to be made, one must know how frequently the attacks stop without simultaneous

5. Mygind, S. H., and Dederding, D.: The Pathogenesis of Ménière's Disease and of Kindred Conditions in the Ear and the Rest of the Body, Ann. Otol., Rhin. & Laryng. **47**:938, 1938; The Diagnosis and Treatment of Ménière's Disease, ibid. **47**:768, 1938; Clinical Experiments with Reference to the Influence of the Water Metabolism on the Ear, ibid. **47**:360, 1938.

6. Furstenberg, A. C.; Richardson, G., and Lathrop, F. D.: Ménière's Disease, Arch. Otolaryng. **34**:1083 (Dec.) 1941.

7. von Frankl-Hochwart, L.: Erfahrungen über Diagnose und Prognose des Ménière-schen Symptomenkomplexes, Jahrb. f. Psychiat. **25**:245, 1905.

impairment or destruction of the function of the internal ear. Statistics of this type are not available. Von Frankl-Hochwart [7] and Furstenberg and associates [8] emphasized the fact that the outlook for hearing was not favorable in their cases. With this in mind one wonders whether cessation of vertigo was due to treatment, to gradual impairment of the function of the labyrinth or to spontaneous cure or remission.

The Vascular Theory.—Ménière propounded the vascular theory. In the past, hemorrhages into the internal ear were considered. The results of microscopic examination in Ménière's syndrome, idiopathic as well as symptomatic, apparently favor the vascular theory. This holds true whether one considers the hydrolabyrinth due to hypersecretion of endolymph or to increased permeability of the blood vessels. The question whether the angioneurotic disturbance consists of a spasm of the internal auditory artery or a dilatation of capillaries with subsequent stasis of the blood cannot be answered definitely. Atkinson [8] expressed the belief that Ménière's syndrome might be due either to vasospasm or to vasodilatation. It is difficult to understand that two distinctly antagonistic mechanisms could produce identical symptoms. Apparently the concept of Atkinson is too schematic. It seems that the angioneurotic disturbance is of a more complicated nature. Nevertheless, clinical experience indicates that angiospasms in Ménière's syndrome are of less importance than in Raynaud's disease, migraine and other genuine vasoneuroses. The following observations support this statement: In hypertension, secondary as well as essential, angiospasms are a common finding. In fact, hypertensive patients frequently complain of vertigo.[9] Yet real Ménière attacks are comparatively rare. Even with angiospasms of the retinal vessels Ménière attacks are not common. In lead poisoning the same is true. Furthermore, vasodilating drugs, such as theobromine, papaverine and atropine, often fail to relieve attacks. Although my experimental observations [10] indicate that dilatation of capillaries is of greater importance than spasm of the internal auditory artery in producing hydrolabyrinth, I do not feel that there is sufficient evidence yet to make a definite statement concerning the causa ultima of the Ménière attack in man.

Most frequently the angioneurotic disturbance within the internal ear is due to arteriosclerosis of the brain. Among 94 cases collected by von Frankl-Hochwart [7] and myself the first attack was observed between the fourth and the fifth decade or later in 51 instances and between the third and the fourth decade in 25 instances. These findings point to cerebral arteriosclerosis as the etiologic factor since arteriosclerotic changes in the internal carotid and the vertebral arteries frequently can be found by microscopic examination in the second decade of life and can always be found after the fourth decade.[11] The frequent combination of Ménière's syndrome with headache, particularly with pain in the occiput, is another finding which points to cerebral arteriosclerosis. The further observation of the patients for a period also supports this concept.[11c] I believe, therefore, that in every case of Ménière's syndrome incipient arteriosclerosis of the brain should be taken into consideration if the first attack takes place between the third and the fifth decade of life and if there is no evidence of another etiologic factor.

The reason for this statement is obvious. The blood vessels of the internal ear originate from the cerebral vessels, which commonly present arteriosclerotic changes. The arteriosclerosis may cause dilatation of the larger vessels and narrowing of the smaller ones. Although the internal auditory artery (similar to the arterioles of the cerebral cortex) seldom shows sclerotic changes, it is obvious that the flow of blood within the internal ear will be disturbed if there are organic changes of the basilar and vertebral arteries. The abnormal blood flow stimulates the abnormal vasomotor reaction of the blood vessels of the internal ear which in turn is responsible for the hydrolabyrinth as well as Ménière's syndrome. The relationship between cerebral arteriosclerosis and angioneurosis of the internal ear is similar to that between arteriosclerosis of the kidney and angioneurosis of the blood vessels of the retina in essential hypertension.

8. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116:** 1753 (April 19) 1941.

9. Furstenberg, A. C.; Maxwell, J. H., and Richardson, G. H.: Essential Hypertension from the Standpoint of Otolaryngology, Tr. Am. Acad. Ophth. **44:**43, 1939.

10. Brunner, H.: Die Pathologie und Therapie der vasomotorischen Erkrankungen des Innenohres, Wien. klin. Wchnschr. **38:**1235, 1925.

11. (*a*) Doerfler, J.: Ein Beitrag zur Frage der Lokalisation der Arteriosklerose der Gehirngefässe mit besonderer Berücksichtigung der Arteria carotis interna, Arch. f. Psychiat. **103:**180, 1935. (*b*) Hiller, F.: Die Zirkulationsstörungen des Gehirnes und Rückenmarkes, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 11, p. 178. (*c*) Stein, C.: Die Arteriosklerose des Gehörorganes, in Alexander, G.; Marburg, O., and Brunner, H.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1928, vol. 2, pt. 1, p. 641.

Accepting this view, one must ask why Ménière's syndrome is not more frequent than it is in cases of cerebral arteriosclerosis. An exact answer cannot be given, but it can be stated that cerebral arteriosclerosis does not cause Ménière's syndrome in every instance probably for the same reason that it does not cause neurologic symptoms in every instance. In fact, cerebral arteriosclerosis is found more often at autopsies than it is diagnosed clinically. Among 400 patients who were found to have cerebral arteriosclerosis at autopsy only 198 had complained of symptoms such as headache, vertigo and loss of memory.[11b] The diagnosis of cerebral arteriosclerosis becomes even more difficult because in these instances sclerosis of the peripheral arteries (except the aorta) is usually absent and the eyeground is usually normal unless the cerebral arteriosclerosis is combined with hypertension or edema of the brain. Thus, Ménière's syndrome, particularly if it is progressive, may be the only symptom of arteriosclerosis of the brain, and it may be followed by other symptoms in a later stage of the disease.

Among the other diseases of the brain that occasionally cause Ménière's syndrome by means of angioneurotic disturbances are tabes and injuries of the head. Tumor of the acoustic nerve is important if small; in the later stage of the disease Ménière's attacks are rare. Cerebellar tumors frequently produce attacks of vertigo but almost never actual Ménière attacks.

Among the systemic diseases various types of leukemia, syphilis, influenza, malaria and virus infection must be considered. While in leukemia bleedings into the internal ear are responsible, the infectious diseases probably exert their influence on the vasomotor nerves of the internal ear as do focal infections. I agree with Wright[12] and others who have stressed the frequency of focal infection as a cause of Ménière's syndrome and the importance of eradicating all accessible foci of infection.

Allergy is occasionally a cause of Ménière's syndrome. In this respect Ménière's syndrome differs from migraine and Quincke's edema, which are frequently caused by allergy. Atkinson[13] expressed the belief that the instances of Ménière's syndrome due to allergy are characterized by the sensitivity to histamine, the presence of which can be proved by an intradermal injection of 0.0057 mg. of a histamine base (0.01 mg. of histamine dihydrochloride). The value of that test is doubted by other investigators.

12. Wright, A. J.: Aural Vertigo, J. Laryng. & Otol. **53**:97, 1938.

13. Atkinson, M.: Diagnosis and Treatment of Ménière's Syndrome, Arch. Otolaryng. **37**:40 (Jan.) 1943.

TREATMENT

As with most diseases in which the etiologic factors are not exactly known, frequent failures of treatment occur. The Ménière attack as such does not require definite treatment since it resolves spontaneously and can be mitigated by such simple procedures as darkening the room, proper position of the patient and administration of bromides and laxatives. Serious injuries in the course of an attack are not frequent.

The treatment of Ménière's syndrome which aims at preservation of the function of the inner ear as well as at prevention of the attacks is still in the experimental stage. At present the following procedure seems to be advisable: First, exclude a Ménière neurosis. The personality of the patient, the lengthy description of the troubles, the permanently normal hearing, the permanent absence of nystagmus and the normal neurologic findings will assist in the differential diagnosis.

If the case is actually one of Ménière's syndrome, the next question is whether the syndrome is idiopathic or symptomatic, instances of the latter being in the majority. If it is symptomatic, the cause may be aural, cerebral or systemic. Among the aural conditions a chronic adhesive process of the tympanic cavity is frequently found.[14] The mechanism by which an adhesive process produces acute hydrolabyrinth is not known. Experience proves, nevertheless, that proper treatment of an adhesive process, particularly in young patients, frequently leads to abatement of Ménière attacks. In older patients the adhesive process is probably not as important as arteriosclerosis of the brain, which in general is the most frequent cause of Ménière's syndrome. The treatment of patients for this factor is difficult because there is no standard therapy for cerebral arteriosclerosis. If the examination does not reveal any pathologic condition of the heart and the kidneys, the general treatment for arteriosclerosis is instituted. Of paramount importance is rest, physical as well as psychic. Hypnotic and sedative drugs are administered. Theobromine combined with calcium should be prescribed for enlargement of the blood vessels and decrease of their permeability. A salt-poor diet similar to the diet of Furstenberg is advisable, specifically on the ground of the unfavorable effect of sodium chloride in all states of abnormal excitability of the blood vessels. Meat in the diet should be

14. Brunner, H.: Ueber akute Schwindelanfälle bei chronisch-adhäsiver Mittelohrentzündung, Monatschr. f. Ohrenh. **57**:317, 1923.

restricted for the same reason. Phobias should be absolutely avoided. The results of the treatment are not uniform; some patients respond well; others, not at all.

In the other types of symptomatic Ménière's syndrome the treatment should be adapted to the etiologic factors.

If no cause can be found, the diagnosis of idiopathic Ménière's syndrome must be made. Cases of the idiopathic type are not common. The treatment is difficult. Specific drugs which influence the sympathetic or the parasympathetic nervous system are not particularly effective. Atkinson [14] has recommended nicotinic acid specifically on the ground that it is a vasodilator, not because it is a vitamin. In his cases the period of observation was too short for him to draw definite conclusions. Schick [15] has advised intravenous injections of a solution of magnesium sulfate. Of 18 patients treated, 14 were cured or improved during a period of only fourteen months. I employ purely symptomatic treatment for patients with idiopathic Ménière's syndrome as for those with arteriosclerosis of the brain. Although I cannot offer exact statistics I do not have the impression of uniform success. To sum up, the conservative treatment of patients with Ménière's' syndrome, symptomatic or idiopathic, is by no means always successful. Surgery [16] therefore has a place. Surgical treatment consisting of destruction of the labyrinth, however, implies certain dangers. In dissection of the vestibular nerve, the cochlear branch can be injured, and there are a few instances of postoperative fatality on record. Operation on the labyrinth [17] as a rule involves the cochlea and the possibility of infection. Although infection can be combated by sulfonamide compounds, the danger to hearing prevents indiscriminate surgical treatment. The indications for surgical intervention in symptomatic and idiopathic Ménière's syndrome are few at present. In my opinion the following criteria should be met: (1) The diagnosis of Ménière's syndrome must be certain; (2) conservative measures must have been given a thorough trial; (3) the patient must be incapacitated by his attacks and unable to attend to his business. In that case the operation certainly should be performed if there is normal hearing in one ear and if the functional test of the other ear at various times proves that the disease is progressive, i. e., if it demonstrates a gradual loss of function in the labyrinth and the cochlea.

It is to be expected that surgical intervention will come into greater favor when a more dependable method of performing partial labyrinthotomy is worked out. In this respect the experiments of Day [18] and Goodyear [19] promise greater success in the near future.

SUMMARY AND CONCLUSIONS

The symptoms of Ménière's syndrome are due to acute hydrolabyrinth.

Acute hydrolabyrinth may resolve without injury to the internal ear or it may lead to fixed dilatation of the inferior part of the internal ear and gradual or acute destruction of the sensorial cells.

The typical Ménière attack consists of tinnitus, diminution of hearing, labyrinthine vertigo and spontaneous nystagmus. Only vertigo of the type which can be produced by the usual clinical methods of examining the labyrinth should be considered as labyrinthine.

Typical Ménière attacks are frequently followed by a Ménière neurosis. This, however, does not present spontaneous nystagmus.

Marked spontaneous nystagmus between Ménière attacks is not of labyrinthine origin; it indicates an organic disease in the posterior cranial fossa.

If the attacks consist only of labyrinthine or only of cochlear symptoms, a definite diagnosis cannot be made unless the patient is under observation for a longer period.

During remission the patient either is normal or, if the injury is progressive, presents gradual diminution of function of the inner ear.

If there is a systemic, cerebral or aural cause the symptom complex is called "symptomatic Ménière's syndrome." When no etiologic factor can be determined, the disease is designated "idiopathic Ménière's syndrome." The pathologic changes are apparently the same in both cases.

Concerning the causes of Ménière's syndrome, the metabolic theory and the vascular theory are offered. The metabolic theory is not supported by microscopic observation of the human ear, by chemical examination of the blood or by treatment. The vascular theory is supported by microscopic observation of the human ear as well as by experiment and explains the pathologic picture of the idiopathic as well as the symptomatic

15. Schick, A.: The Treatment of Ménière's Syndrome with Magnesium Salts, Ann. Otol., Rhin. & Laryng. **52**:45, 1943.

16. Dandy, W. E.: The Surgical Treatment of Ménière's Disease, Surg., Gynec. & Obst. **72**:421, 1941.

17. Tobey, H. G.: Medical Treatment of Ménière's Disease, Surg., Gynec. & Obst. **72**:425, 1941.

18. Day, K. M.: Labyrinth Surgery for Ménière's Disease, Laryngoscope **53**:617, 1943.

19. Goodyear, H. M.: Ménière's Disease, Laryngoscope **53**:743, 1943.

Ménière's syndrome, although further studies are necessary in this respect.

Most frequently the angioneurotic disturbances within the internal ear are due to arteriosclerosis of the brain. Incipient arteriosclerosis of the brain should be taken into consideration in any case of Ménière's' syndrome in which the first attack takes place between the third and the fifth decade of life and there is no evidence of another cause such as tabes, injury of the head or small tumor of the acoustic nerve.

Among the systemic diseases, various types of leukemia, syphilis, influenza, malaria, virus infection and particularly focal infection may cause the symptomatic Ménière's syndrome.

Allergy is occasionally but not frequently a cause of the symptomatic Ménière syndrome.

Among the aural diseases, a chronic adhesive process of the tympanic cavity is frequently found to cause the symptomatic Ménière syndrome.

Concerning treatment, a Ménière neurosis must be excluded. When Ménière's syndrome is symptomatic, the etiologic disease should be treated. There is no satisfactory treatment for the idiopathic Ménière's syndrome. Surgical intervention is indicated if the diagnosis of Ménière's syndrome is certain, if conservative measures have been exhausted and if the disease is progressive on one side while the other ear is normal.

307 North Michigan Boulevard.

ABSORPTIVE CAPACITY OF THE NOSE

MAURICE SALTZMAN, M.D.
PHILADELPHIA

This study concerns the absorptive ability of the respiratory portion of the mucous membrane of the nose as it applies to nasal medication and immunization. As there is a paucity of experimental data on this subject, my deductions will be based on clinical observations as appraised in the light of the new concepts of physiology.

It seems that prescribing physicians have little fear that an untoward effect might occur from the use of a sympathomimetic drug in the nose. The universal employment of nose drops containing ephedrine is significant. However, from time to time there appears in the literature a report of a toxic reaction from a nasal treatment with a vasoconstrictor. Greene and Greenspan [1] observed restlessness in children following the use of nose drops containing ephedrine: In a group of 19 normal infants and young children who were given drop doses of a 1 per cent aqueous solution of ephedrine sulfate by intranasal instillation, 8 manifested irritability and 4 insomnia. Lapin and Weichsel [2] reported a case of ephedrine poisoning in a 5 month old infant, manifested by extreme nervousness and paroxysms of muscular contractions, after the use of a 3 per cent solution of ephedrine sulfate as nose drops. The instillation of ephedrine nose drops was found by Powers [3] to be a cause of uterine bleeding, through the medium of pituitary stimulation, and Zeller [4] reported a case of dermatitis venenata resulting from the use of ephedrine intranasally. O'Donnell [5] reported 2 cases of a toxic reaction with alarming symptoms from the treatment of sinusitis with paredrine-sulfathiazole suspension [6] by the displacement method. The symptoms were headache, palpitation, vomiting and sensations of chilliness in the hands and legs. The onset occurred soon after the treatment, and the duration of the severe symptoms was several hours. The reaction to a sympathomimetic drug gives all these features. In these cases paredrine was undoubtedly responsible; the sulfathiazole caused the viscosity in the mixture, the suspension of microcrystals, soaked in paredrine, clinging tenaciously to the sinus walls and enhancing the absorption of the vasoconstrictor drug. Every rhinologist has frequently enough been caused some anxiety when the patient began perspiring and complained of palpitation of the heart after the nasal mucosa had been shrunk with pledgets of cotton saturated with a solution of ephedrine sulfate, neo-synephrine hydrochloride or a similar substance. As the patient recovers fairly promptly following such an episode, the specialist develops an attitude of complacency toward these symptoms.

That the topical application of a vasoconstrictor to the nasal mucosa is considered a safe procedure ordinarily is due to the fact that no systemic reaction is anticipated since absorption from the mucous membrane of the nose is said to be taking place very slowly. The experiments of Childrey and Essex [7] bear out this assumption. No appreciable effect on the blood pressure took place when 50 mg. of ephedrine was deposited in the frontal sinus of a dog, whereas the subcutaneous injection of a similar quantity of ephedrine caused a rise in blood pressure lasting as long as sixty minutes. Similar experiments were carried out by these authors with other highly active substances, such as histamine, rattlesnake

From Temple University School of Medicine and the Mount Sinai Hospital.

Read at the joint meeting of the Section on Otolaryngology of the College of Physicians and the Philadelphia Laryngological Society, Nov. 17, 1943.

1. Greene, D., and Greenspan, L.: Undesirable Effects of Intranasal Use of Ephedrine in Children: Preliminary Report, Dis. Eye, Ear, Nose & Throat **2**:251-253 (Aug.) 1942.

2. Lapin, J. H., and Weichsel, M.: Unusual Case of Ephedrine Poisoning, J. Pediat. **15**:722-723 (Nov.) 1939.

3. Powers, E. G.: Ephedrine Sulphate Intranasally as Cause of Uterine Bleeding, Texas State J Med. **37**:369-372 (Sept.) 1941.

4. Zeller, M.: Hypersensitivity to Ephedrine and Ephetonine, J. A. M. A. **101**:1725-1726 (Nov. 25) 1933.

5. O'Donnell, M. C.: Toxic Reactions of Paredrine-Sulfathiazole: Report of Two Cases, J. A. M. A. **122**: 298 (May 29) 1943.

6. Paredrine-sulfathiazole suspension is a preparation introduced by the Smith, Kline and French Pharmaceutical Company. It is a 5 per cent suspension of sulfathiazole microcrystals in an isotonic solution of 1 per cent paredrine hydrobromide (p-hydroxy-a-methylphenylethylamine hydrobromide). The preservative used is sodium ethylmercurithiosalicylate, 0.005 per cent; gelatin is present in the proportion of 0.1 per cent.

7. Childrey, J. H., and Essex, H. E.: Absorption from the Mucosa of the Frontal Sinus, Arch. Otolaryng. **14**:564-569 (Nov.) 1931.

venom, phenolsulphonphthalein, nicotine and epinephrine, and it was found that the absorption from the nasal cavities and the sinuses was much slower than that from the subcutaneous tissue.

During the winter of 1942-1943 I had occasion to treat 14 children aged 10 to 17 years, whose normal systolic pressure ranged between 104 and 120. These children were all suffering from acute infections of the upper respiratory tract, superimposed on hyperesthetic rhinitis. The inferior turbinates of these children were so congested that they pressed on the septum and there was complete obstruction to breathing. In 6 children the anterior part of the septum was deformed to the extent that only the smallest of cotton-tipped probes could be inserted into the constricted side. To relieve the obstruction to nasal breathing, pledgets of cotton saturated with 0.25 per cent neo-synephrine hydrochloride were inserted, by means of a wooden applicator, between the inferior turbinate and the septum. Good results were obtained in the 8 children whose septums were not markedly deviated to one side. Only 1 patient complained of slight headache following the treatment, and his systolic pressure rose to 120, whereas his previously recorded reading was 112. The headache, slight nausea and sweating lasted about an hour. However, the 6 patients who had badly deformed septums obtained no relief from this weak solution on the stenosed side. Accordingly, a cotton-tipped wooden applicator moistened with a 1 per cent solution of neo-synephrine hydrochloride was inserted between the inferior turbinate and the septum on the constricted side with a minimal amount of pressure. Good shrinkage was obtained in about five minutes in all the patients, but a toxic reaction followed in 1. This patient, a boy aged 14 years, was seized with a severe occipital headache and a feeling of stiffness of the back of his neck. His systolic pressure, which was 104 prior to the treatment, rose to 130. Vomiting and profuse sweating followed, the subsidence of symptoms taking place gradually over a period of two hours. When seen eight hours later, the patient complained of weakness, but he was otherwise symptom free. His systolic pressure was 110.

It appears that a toxic reaction which occurs when a sympathomimetic drug other than epinephrine has been absorbed by the nasal route lasts several hours. Epinephrine, which is a dihydroxybenzene, is destroyed in the body within a few minutes following its absorption, whereas the monohydroxybenzenes, paredrine and neo-synephrine, produce lasting effects. Rapid oxidation of epinephrine in the tissues takes place, but this fate is not shared by the other sympathomimetic drugs. It was formerly held that the enzyme amine oxidase brings about the destruction of epinephrine in the body. Richter and Tingey[8] pointed out that amine oxidase is primarily an intestinal enzyme and that it is not a rapidly acting substance. Paredrine and neo-synephrine are amines and would have been acted on by amine oxidase if that enzyme were capable of bringing about rapid oxidation. The sympathomimetic amines differ in stability, and it is only epinephrine which is speedily oxidized in the body, by a process not yet understood.

The absorptive ability of the nasal mucous membrane cannot be appraised unless one reviews first the newer concepts of the physiology of the nose, especially those concerning the cilia and the blanket of mucus covering the posterior two thirds of the respiratory mucosa. The cilia beat at the rate of 250 impulses a minute and propel the mucous blanket toward the nasopharynx and esophagus. As the mucus keeps on moving toward the stomach, the nasal glands secrete and replace a new blanket of mucus every ten minutes. It follows therefore that a solution instilled into the nose by means of a dropper will fall on a glossy slippery protective covering which is in constant motion. Little can be absorbed by the underlying epithelial cells under these circumstances. However, if the solution contains a drug which paralyzes the cilia, causing the mucous covering to become stationary, a greater amount of penetrability and absorption can be expected. Cocaine in concentration of 5 per cent or over is an example. Likewise, if the substance instilled into the nose is a solvent of mucin, the protective effect of the mucous blanket will be eliminated. Highly alkaline or highly acid solutions act in this manner. The same reasoning holds true in regard to the sinuses. The blanket of mucus extends into the sinuses, and the cilia there are as active as in the respiratory portion of the nasal cavities. It is different, however, in the anterior third of the nasal cavity, where the cilia are scanty and the mucin drains sluggishly by gravity; here the mucosa will possess an absorptive capacity approaching that of any other mucous membrane of the body. Regarding the sinuses, however, the physical force of viscosity must be taken into consideration. According to Proetz,[9] the prop-

8. Richter, D., and Tingey, A. H.: Amine Oxidase and Adrenaline, J. Physiol. **97**:265-271 (Dec. 14) 1939.

9. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Publishing Co., 1941, p. 64.

erty of viscosity is of paramount importance in the displacement treatment of the sinuses. When a solution of low viscosity is instilled into a sinus, the emptying time is about one-half hour. On the other hand, if the substance introduced is iodized oil, it will generally take ninety-six hours to leave the sinus completely. When a solution of low viscosity is forced into a sinus by the displacement process, the cilia and the moving blanket of mucin will hinder absorption the same as in the nasal cavity. On the other hand, if the substance which has entered the sinus is tenacious, it will be retained in the cavity over a longer period and absorption will be facilitated. Moreover, the reason why a patient is given a displacement treatment by the Proetz method is because one of his sinuses contains stagnant secretions, the cilia being inactive and the epithelial lining being inflamed or pyogenic. Under these conditions the capacity for absorption by the mucous membrane will be altered from the physiologic normal.

Selectivity as to which substance is to be readily absorbed plays an important role in the function of the mucous membrane of the nose. Blumgart [10] pointed out that pituitary extract is rarely effective when administered orally or rectally but that it is of great therapeutic value when absorbed intranasally. Kintner and Greene [11] reported that their patients with diabetes insipidus had an average daily urinary output of 9,550 cc. When 1 cc. of a solution of pituitary was injected subcutaneously, the output was only 3,700 cc., while the administration of the pituitary solution by nasal spray reduced the urinary output to 4,100 cc. On the other hand, when the same quantity of solution of pituitary was administered by nasal swab, the urinary output amounted to 5,400 cc. In obstetrics Hofbaur [12] found that labor was induced by inserting a tampon saturated with pituitary solution between the inferior turbinate and the septum. The reason for the rapid absorption from this area, according to Hofbaur, is that the blood and lymph channels of the mucous membrane covering the inferior turbinate are markedly increased during pregnancy. Nevertheless, in atrophic rhinitis the mucosa with its reduced blood supply, hyalinized subcutaneous connective tissue and stratified squamous epithelium constitutes a suitable absorbing surface for estrogenic substance. In this instance it is the dry soil that imbibes moisture most readily. The rationale of this form of therapy is based on the theory of the nasogenital relationship. Anatomic sites in the nasal mucosa, localized by Fleiss [13] on the ventral portion of the inferior turbinate and the tuberculum septi, were designated the "genital spots." Mortimer, Wright and Collip [14] in a study of the cranial roentgenograms of 68 patients with atrophic rhinitis and ozena found evidence of pituitary dystrophy in the large majority of them. On an endocrinologic basis, these authors advocated as specific therapy for atrophic rhinitis the local application of estrogenic substance to the mucosa. The rationale for the local treatment of senile vaginitis with estrogen is closely akin to that for the treatment of atrophic rhinitis with estrogen, and the same analogy is applicable to all the "sex skin" areas. In a histologic study of the vaginal epithelium following estrogenic therapy, Davis [15] found that the basal cells, which were formerly inactive, were proliferating and showing abundant mitosis; an increase in the glycogen content of the cells was also noted. These results from estrogenic therapy in degenerated tissues are amazing and the absorptive ability of the mucosa in atrophic rhinitis and senile vaginitis exemplifies selectivity, the lining membrane appropriating the hormones by virtue of its sex relationship.

In discussing the absorptive ability of the mucosa of the nose and sinuses, Childrey and Essex [7] in 1931 quoted the contradictory views of numerous authors on the role of the upper air passages as foci of infection. The inference was that if frequently no severe systemic reaction resulted from infection of the sinuses, the absorptive capacity of these cavities must be limited. With the evolvement of the doctrine of the presence of lysozymes in the tissue fluids the view is now held that together with the phagocytic cells the antibodies of the nasal secretion constitute a potent defense mechanism. The presence of lysozyme in mucin has been demonstrated by

10. Blumgart, H. L.: Antidiuretic Effect of Pituitary Extract Applied Intranasally in a Case of Diabetes Insipidus, Arch. Int. Med. **29**:508-514 (April) 1922.

11. Kintner, A. R., and Greene, C. H.: Administration of Pituitary Extract by Nasal Spray in Treatment of Diabetes Insipidus: Atomizer Modified for Use of Small Amounts of Liquid, J. A. M. A. **91**:1370-1371 (Nov. 3) 1928.

12. Hofbaur, J. I.: Nasal Application of Solution of Pituitary for Obstetric Purposes: Preliminary Communication, J. A. M. A. **89**:24-25 (July 2) 1927.

13. Fleiss, cited by Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920, p. 300.

14. Mortimer, H.; Wright, R. P., and Collip, J. B.: Atrophic Rhinitis: Constitutional Factor and Treatment with Estrogenic Hormones, Canad. M. A. J. **37**:445-456 (Nov.) 1937.

15. Davis, M. E.: Treatment of Senile Vaginitis with Ovarian Follicular Hormones, Surg., Gynec. & Obst. **61**:680-686 (Nov.) 1935.

Daly.[16] Recent research indicates the presence in the nasal secretions of specific inactivating agents against the viruses of poliomyelitis and epidemic influenza. While the nose harbors various pathogenic bacteria, their penetration into the deeper tissues is prevented most often by the antibodies on the surface, which destroy them, and by the moving blanket of mucus, which removes them. Moreover, it may be assumed that the killed bacteria act as antigens, stimulating the production of new antibodies. That some of these defensive products enter the system by a process of selective absorption was shown by Walsh and Cannon,[17] who found specific agglutinins in the blood and various tissues of the rabbit after the intranasal instillation of a vaccine containing Bacterium typhosum and Bacterium paratyphosum B. These authors demonstrated, however, that the antibodies formed locally remained in higher concentration in the nasal tissues than elsewhere in the body. The localizing character of this defense mechanism was further shown by Walsh and Cannon,[17] who after repeated instillation of a vaccine into the nostrils of a rabbit found an accumulation of leukocytes, macrophages and mononuclear cells in the nasal tissues.

Whether the results obtained from nasal vaccination in the rabbit can be duplicated in man is a question to be determined by further clinical investigation. Vaccination by the subcutaneous route against infections of the upper respiratory tract has had an extensive clinical trial over a period of three decades, but no evidence has been gathered which proves that colds were prevented by this form of immunization. In 1918 Eyre and Lowe[18] reported the results of vaccination against colds in 1,000 troops as compared with another 1,000 nonimmunized soldiers who served as controls. There was an incidence of colds of 1.2 per cent in the treated groups, compared with 7.5 per cent in the untreated controls. On the other hand, Ferguson, Davey and Topley[19] concluded that prophylactic vaccination against colds was useless. This conclusion was reached after a study of a series of 138 treated patients and 148 controls. In 1937 Houser[20] reported his results from prophylactic vaccination against colds in a group of 86 students. The only benefit noted was a decrease in the severity and the duration of the disease; its incidence was not lessened. On the other hand, in 1938 Sherman[21] found, in a large industrial practice after an extensive trial calculated on the efficiency of the employees, that no benefit whatever was derived from subcutaneous vaccination relative to reduction in the number, the severity or the duration of colds. Walsh[22] reported favorable results from intranasal vaccination in 74 per cent of his cases. The reported occurrence of pneumonia following intranasal vaccination in laboratory animals served as a deterrent to the employment of this method of immunization in human subjects. Weiss,[23] however, is convinced that the epiglottis in man is so shaped as to form a perfect barrier against the entrance into the larynx of droplets coming down from the nose. As a further precaution against aspiration pneumonia, he advised that the antigenic matter introduced into the nose be limited to one drop or one spray.

Freedom from, or susceptibility to, nasal disease is largely influenced by individual human elements. The human nose is subject to trauma at birth, and beatings are perpetually inflicted on it during the growth period of childhood and adolescence. Asymmetry of the septum and deformities of the external nose bring about overventilation in one nostril and stenosis in the other. Hilding[24] demonstrated morphologically that the epithelium will be radically altered under these circumstances: In the overventilated nostril the epithelium will change to the stratified squamous type and the cilia will become inactive, and in the stenosed side mucus-filled goblet cells without cilia will become numerous. Rickets that makes its appearance in infancy is deleterious to both the nasal framework and the lining membrane, and deficiencies of vitamins A and C are manifested in vulnerable epithelium. Glandular disturbances and cardiac disease affect nasal

16. Daly, S.: Lysozyme of Nasal Mucus: Method of Preparation and Preliminary Report on Its Effect on Growth and Virulence of Common Pathogens of Paranasal Sinuses, Arch. Otolaryng. **27**:189-196 (Feb.) 1938.

17. Walsh, T. E., and Cannon, P. R.: Immunization of Upper Respiratory Tract, Arch. Otolaryng. **20**:820-836 (Dec.) 1934.

18. Eyre, J. W. H., and Lowe, C. E.: Prophylactic Vaccination Against Catarrhal Affections of the Respiratory Tract, Lancet **2**:484-487 (Oct. 12) 1918.

19. Ferguson, F. R.; Davey, A. F. C., and Topley, W. W. C.: Value of Mixed Vaccines in Prevention of Common Cold, J. Hyg. **26**:98-109 (March) 1927.

20. Houser, K. M.: Analysis of Results of Vaccination of College Students Against Colds, Arch. Otolaryng. **26**:283-290 (Sept.) 1937.

21. Sherman, J. B.: Prophylaxis of Common Cold, Brit. M. J. **2**:903-904 (Oct. 29) 1938.

22. Walsh, T. E.: Intranasal Vaccine Spray: Its Use in Prophylaxis Against Common Cold, Ann. Otol., Rhin. & Laryng. **49**:875-894 (Dec.) 1940.

23. Weiss, E.: Spreading of Vaccine in Nasal Vaccination, Arch. Otolaryng. **33**:623-630 (April) 1941.

24. Hilding, A.: Experimental Surgery of the Nose and Sinuses: I. Changes in the Morphology of the Epithelium Following Variations in Ventilation, Arch. Otolaryng. **16**:9-18 (July) 1932.

function profoundly. The delicate vasomotor system in man gets out of balance through overindulgence in food, drink and sexual stimulation and the "stuffy nose" becomes evident. Man is prone to allergic disorders. The histamine-like substance in the nasal tissue is released by the injury of the specific antigens, causing an increase in the permeability of the capillary blood vessels and a copious extravasation of serum. The edema produced by allergy will lead to immobilization of the cilia, stagnation of the secretion and secondary infection. The rhinorrhea of hay fever, hyperesthetic rhinitis and the "virus cold" will wash away the highly defensive nasal secretions with their specific lysozymes, and the hydrogen ion concentration will become alkaline. The significance of the hydrogen ion concentration in the nasal secretion has been fully described by Fabricant,[25] who expressed the belief that the bacteria producing infectious rhinosinusitis grow best in an alkaline medium. The warm air and low humidity of the average bedroom in the winter produce a drying effect on the mucous blanket and slow the movement of the cilia. In his daily work man often breathes smoke-laden air that turns his nasal mucin into black paste. At other times the nasal tissues are exposed to freezing temperature with its congesting effect. Exposure of the nasal tissues to rarefied air in airplane travel is another prevalent cause for disturbed nasal function.

Absorption will be increased in the overventilated nostril in which the mucin dries rapidly and the movement of the cilia is slow. Likewise, the breathing of dry warm air will immobilize the cilia, reducing their efficiency as sweepers; a greater amount of absorption will take place in the underlying epithelium. The increased blood supply in nasal tissues congested from breathing cold or rarefied air or from vasomotor disturbance will enhance their absorptive capacity. In rhinorrhea a substance introduced into the nose will be washed away so rapidly that absorption will be prevented. Moreover, in allergy a vasoconstrictor administered intranasally will be neutralized by the dilative effect of the histamine-like substance on the blood vessels in the nasal tissues.

25. Fabricant, N. D.: Significance of the p_H of Nasal Secretion in Situ, Arch. Otolaryng. **34**:150-163 (July) 1941.

SUMMARY AND CONCLUSIONS

Besides having the functions of olfaction and respiration, the nasal mucous membrane constitutes a highly specialized mechanism of defense, consisting of mechanical, immunologic and phagocytic components. The absorptive capacity of the nasal mucosa is variable. An anatomic deformity will affect this capacity quantitatively, while a pathologic condition will modify it both as to amount and as to effect. Absorption of medicines by the lining membrane of the nose depends on the method of administration, less absorption taking place when they are administered by nasal swab than when they are introduced through an atomizer. A medicated tampon inserted between the inferior turbinate and the septum brings about a high amount of absorption. The absorptive capacity of the mucous membrane of the nose and the sinuses is inversely proportional to the efficiency of the "sweepers"—the cilia and the mucous blanket—of these cavities. An increase in the blood and lymph circulation of a portion of the nasal mucous membrane or of a sinus, whether of physiologic or pathologic cause, will enhance the absorptive capacity of that surface. When a medicine is absorbable, the degree of its absorption in a sinus after it has been introduced there by the displacement method is proportional to its viscosity. The longer it is retained in the sinus, the more the absorption that will take place. Selectivity is a feature of the absorptive capacity of the nasal mucous membrane. By selective action it readily absorbs the endocrine hormones of the pituitary and the ovaries. Specific antigenic matter when sprayed into the nose brings about a multiplicity of phenomena. Phagocytes concentrate in the tissues, while on the surface antibodies are formed. Some antibodies are absorbed into the general system, but a greater concentration is stored locally. Intranasal vaccination deserves further clinical investigation. The individual etiologic factors—constitutional and environmental—must receive primary consideration.

529 Snyder Avenue.

CANCER OF THE TRACHEA

REPORT OF FIVE CASES

GILBERT E. FISHER, M.D.
BIRMINGHAM, ALA.

Recent reviews of the literature reveal that cancer of the trachea is rare. Some 60 cases of primary cancer of the trachea were reported up to 1924; in 1933 the number of cases has increased to 154.[1] The first recorded observation ot a tracheal new growth was that of Lieutaud in 1767.[2] Semon stated that the frequency relation of laryngeal to tracheal tumors was 100: 1.[2]

As can be readily seen from the accompanying table, carcinoma is found much more frequently than is sarcoma.

Primary carcinoma of the trachea usually arises in the lower third of the trachea and is of the squamous cell type. It is a highly malignant growth which develops insidiously. The initial symptom is usually an irritating cough, which

Number of Cancers of the Trachea According to Different Reviewers (After D'Aunoy and Zoeller[3])

	von Bruns, 1898	Krieg, 1908	Lombard and Baldenweck, 1914	D'Aunoy and Zoeller, 19-9
Sarcoma.....	14	21	23	26
Carcinoma...	31	40	54	91
	45	61	77	117

may or may not be accompanied by blood-streaked sputum. This growth has a broad base, and as it increases in size dyspnea is produced, which may be sufficient to cause suffocation unless the patient is relieved surgically. Ulceration of the surface of the growth may result in fatal hemorrhage.

The diagnosis of a tracheal neoplasm can be made only by direct tracheoscopy and removal of a piece of the tissue for microscopic study.

Carcinoma of the trachea appears to spread by direct involvement of the surrounding mucosa and may perforate the posterior wall at an early stage. It metastasizes late in the course of the disease.

There seems to be only one satisfactory type of treatment in these cases. This consists of electrocoagulation of the growth through the bronchoscope with the tumor under direct vision. The electrocoagulation should be followed by irradiation of the tumor with roentgen rays of high voltage. The electrocoagulation can be repeated as necessary to afford adequate space in the tracheal lumen for respiration. The first patient encountered by me and treated by the method just described was seen in the bronchoscopic clinic of the Johns Hopkins Hospital. His case was reported in an earlier publication by Dr. E. N. Broyles and myself.[4] He lived ten years from the time he was first treated.

In this report I present 5 cases of cancer of the trachea seen in private practice during the past three years.

REPORT OF CASES

CASE 1.—Mr. M. R. H., a white man aged 43, was first seen in consultation Oct. 28, 1940. He complained that he had experienced dyspnea and wheezing on inspiration and expiration for the past eight months. For the past two months there had been periodic irritating coughing paroxysms, accompanied by bloody sputum. A constant dull substernal aching sensation was present. There had been no appreciable loss of weight. A roentgen examination of the chest revealed no pathologic condition.

On November 4 a 9 mm. bronchoscope was passed with the patient under local anesthesia and a large mulberry-like growth was found attached to a wide base on the posterior tracheal wall halfway between the larynx and the bifurcation of the trachea. This growth filled approximately 85 per cent of the tracheal lumen and bled freely on manipulation. A piece of tissue was taken for microscopic study. This proved to be adenocarcinoma.

On November 18 a 7 mm. bronchoscope was passed and the growth was destroyed as thoroughly as possible by electrocoagulation. This procedure was repeated

Read before the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 8, 1944.

1. Thomson, St. C., and Negus, V. E.: Diseases of the Nose and Throat, ed. 4, New York, D. Appleton-Century Company, Inc., 1937, p. 655.

2. Cited by D'Aunoy and Zoeller.[3]

3. D'Aunoy, R., and Zoeller, A.: Primary Tumors of the Trachea, Arch. Path. **11**:589-600 (April) 1931.

4. Broyles, E. N., and Fisher, G. E.: Bronchoscopic Experiences with Lung Tumors, Surgery **7**:918-923 (June) 1940.

Dec. 8 and Jan. 5, 1941. No bleeding occurred during the interim.

On February 4 the patient was seen by Dr. E. N. Broyles, of Baltimore, and under direct tracheoscopy the growth was given a seven minute exposure to 1,268 millicuries of radon. It was given a similar exposure to radon six weeks later. Following this treatment, he was vastly improved; his dyspnea disappeared, and he was able to carry on his occupation as a grocer.

On April 26, 1942 bronchoscopic examination revealed that the tumor filled about 40 per cent of the tracheal lumen. The growth did not bleed freely. It was treated by electrocoagulation. This was followed by two series of treatments with high voltage roentgen rays.

He was last seen on July 6, 1943 and appeared to be in good health. He did not complain of weakness, dyspnea or pain and was working daily.

It is of interest that the niece of this patient came to me March 31, 1941, complaining of cough and hemoptysis. She was a 17 year old student. Roentgen examination of the chest revealed no pathologic condition. A 7 mm. bronchoscope was passed, and a bleeding point was found on the posterior tracheal wall approximately halfway between the larynx and the bifurcation. No ulcer, growth or varix could be seen. The bleeding point was treated with electrocoagulation. No further bleeding occurred until August 10, when she again experienced hemoptysis. Bronchoscopic examination revealed active bleeding from the site previously treated. This was again treated by electrocoagulation. Bleeding began again July 25, 1942, and this same procedure was carried out July 29. She was given a series of treatments with high voltage roentgen rays and has been in good health since then.

CASE 2.—Mr. B. P. A., a white man aged 60, was first seen on May 14, 1943, complaining of chronic cough, dyspnea and wheezing of approximately one year's duration. He had become progressively weaker, lost weight and experienced substernal pain. There had been no hemoptysis. Roentgen examination of the chest revealed no pathologic condition. He was advised to have a bronchoscopic examination, but this was refused.

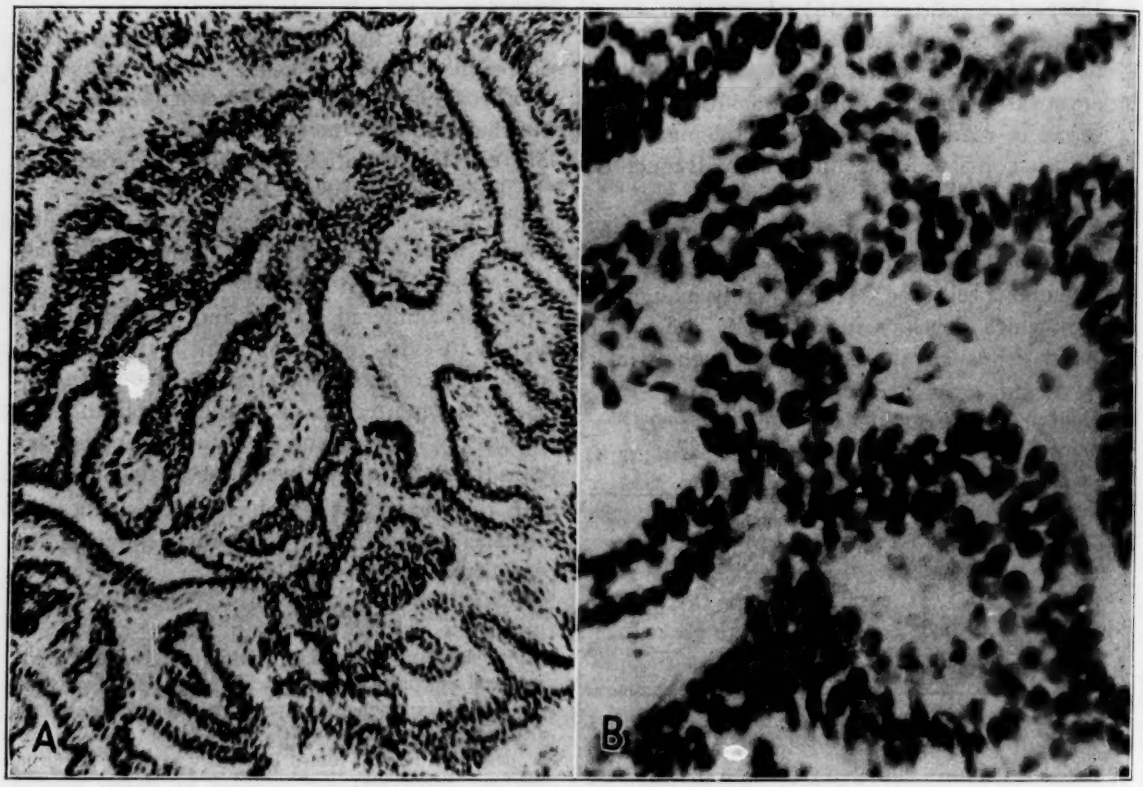


Fig. 1 (case 1).—*A*, low power magnification. *B*, high power magnification. Section through the tumor shows a papillary type of growth made up of irregular masses of blue-staining cells with a loose and in some places edematous stroma. In these papillary and sometimes cystic spaces the lining consists of two layers of cuboidal cells with dark-staining oblong outer limiting membrane. In one part of the tumor there are small cystic spaces partially filled with pink-staining, slightly refractile amorphous material. In this area the double layer of cells lining the cyst is less obvious.

He returned on October 25 in a seriously weakened condition, and bronchoscopic examination was carried out the same day. It revealed a large tumor filling approximately 75 per cent of the tracheal lumen just above the bifurcation of the trachea with extension into the left bronchial lumen. A piece of tissue was taken for microscopic examination. This revealed epithelioma grade III, already invading the lymphatics. On October 28 a large painful lymph gland was palpable above the left clavicle and a very tender spot was found just above the lower end of the right radius. Roentgen examination of the bones of the forearm revealed a bony metastasis at this point. Since that date he has been treated with high voltage roentgen rays.

CASE 3.—Mr. M. J. A., a white man aged 50, a farmer, was seen in consultation by me on March 25, 1943. He complained of dyspnea and wheezing of five months' duration. He had noted cough and blood-streaked sputum for the past three months. There had been no substernal pain or loss of weight.

A 7 mm. bronchoscope was passed with the patient under local anesthesia on the date mentioned. In the lower third of the trachea a large bleeding growth, with an ulcerated surface, filled approximately 75 per cent of the tracheal lumen. This growth had a broad base and was attached to the posterior tracheal wall. A small piece of tissue was removed. Microscopic examination revealed squamous cell carcinoma. High voltage irradiation of the growth was started the following day. This produced a lively improvement in his condition. Bronchoscopic examination June 29 revealed that the growth filled approximately 15 per cent of the lumen of the trachea. It was fulgurated, with electrocoagulation, and a second course of treatment with high voltage roentgen rays was instituted. He was seen one week ago and seemed to be in good physical condition.

CASES 4 and 5.—Two other patients with squamous cell carcinoma of the trachea have been seen by me during the past two years. Both presented far advanced lesions which practically filled their tracheal lumens. One of these patients had complete obstruction of the esophageal lumen at the level of the tracheal lesion. This patient died ten days after the original endoscopic examination. The other patient died one month after endoscopic examination and biopsy.

Fig. 2 (case 3).—Section through the specimen shows the surface covered by a ciliated respiratory type of epithelium. Directly beneath this are broad infiltrating cords of epithelium, the individual cells of which are hyperchromatic and vary somewhat in their size and shape. Mitoses are frequently encountered. There is little tendency to pearl formation, and almost no keratohyalin is being produced.

CONCLUSIONS

1. Cancer of the trachea is rare.

2. Carcinoma is found more frequently than sarcoma.

3. Fulguration of the growth, with electrocoagulation, through the bronchoscope, followed by irradiation of the lesion with high voltage roentgen rays, seems to be the treatment of choice at the present time.

The treatment with high voltage roentgen rays was given by Drs. J. A. Meadows and Karl Kesmodel, of Birmingham, Ala.

501 Medical Arts Building.

PREVENTION OF TRAUMATIC DEAFNESS

FURTHER STUDIES

CAPTAIN WILLIAM H. WILSON
MEDICAL CORPS, ARMY OF THE UNITED STATES

The rapid industrial development in the United States has not been met by an adequate attempt to prevent resultant increase in traumatic deafness in the noisier branches of industry. This is due largely to the employers' refusing to recognize the entity for fear of raising the question of compensation, to the employees' evading the subject lest employment be refused those in whom hearing impairment appears and to a general lack of interest from both the legislative and the scientific points of view.

That compensation boards are making awards for losses of hearing was observed by Bunch.[1] To determine how recent is this trend questionnaires were sent to the compensation commissions of each state. The commissions were asked whether the compensation laws of their state recognized occupational deafness as compensable; whether such recognition, if given, was statutory or based on decisions in the courts of the state, and at what time the state first recognized occupational deafness as compensable. The commissions were also asked to give the approximate number of persons who had received compensation under such a law and to state whether occupational deafness was on the increase or the decrease in their particular state.

In compiling the material from this questionnaire it became evident that occupational deafness as such was recognized as an occupational disease. Deafness that developed over a period as a result of exposure to loud noises and vibrations fell in this category. In contrast, deafness resulting from an accident arising out of and in the course of employment was taken as something quite different. This differentiation has also been made by Reed and Emerson.[2]

While twenty-three states recognized deafness as compensable if it was the result of an accident, only nine considered it compensable as an occupational disease. Three of the nine had enacted a law making it such in 1943, one in 1941 and two in 1940. Here the trend was recognizable. Six states admitted that if deafness could be proved to be disabling it might be compensable because of that, but not in and of itself. Nine states reported that occupational deafness was not recognized as compensable, but no information was furnished relative to deafness resulting from accident. One state reported that there was no workman's compensation law.

All states considering occupational deafness compensable as an occupational disease reported such recognition as statutory. Only Arkansas could furnish figures as to the number of persons who had received compensation under such a law. Six persons had been compensated in a three year period. No state was able to furnish information as to whether occupational deafness was on the increase or the decrease in their particular state.

TABLE 1.—*Grouping of States According to Recognition of Occupational Deafness in Their Compensation Laws*

States in which compensation laws recognize occupational deafness as compensable, with date of enactment:

Alabama, 1940	Nebraska, Aug. 29, 1943
Arkansas, Dec. 5, 1940	Oregon, July 1, 1943
California, August 1915	Washington, June 10, 1941
Connecticut	Wisconsin, 1919
Minnesota, 1943	

States in which occupational deafness may be compensable only because of its being disabling, but not in and of itself:

Louisiana	Michigan	Ohio
Massachusetts	New York	Rhode Island

States in which deafness is compensable only when it is the result of an accident arising out of and in the course of employment:

Colorado	Kentucky	North Dakota
Delaware	Maryland	South Dakota
Florida	Missouri	Tennessee
Georgia	Nevada	Texas
Idaho	New Hampshire	Vermont
Illinois	New Jersey	West Virginia
Indiana	New Mexico	Wyoming
Iowa	North Carolina	

States in which occupational deafness is not recognized as compensable, no information being furnished relative to deafness resulting from accident:

Arizona	Montana	South Carolina
Kansas	Oklahoma	Utah
Maine	Pennsylvania	Virginia

State having no workmen's compensation law: Mississippi

The solution to the problem of traumatic deafness lies in its prevention. In discussing deafness of aviators, Bunch[3] stressed the different

1. Bunch, C. C.: Conservation of Hearing in Industry, J. A. M. A. **118**:588-593 (Feb. 21) 1942.

2. Reed, J. V., and Emerson, C. P.: The Relation Between Injury and Disease, Indianapolis, Ind., The Bobbs-Merrill Company, 1938, p. 536.

3. Bunch, C. C.: The Problem of Deafness in Aviators, War Med. **1**:873-886 (Nov.) 1941.

degrees to which pilots are affected. He suggested that further studies be made to determine "whether certain candidates should be excused from this type of training because they are more susceptible than others to the effects of noise."

In a previous paper,[4] the importance of the prevention of traumatic deafness was stressed, and a means of screening out susceptible persons was presented. A possible later simplification of the procedure was suggested. It was with this in mind, in addition to the further establishment of the practicability of the method, that experimental studies were carried out.

The subjects for these studies were enlisted personnel stationed at a quartermaster replacement training center for their basic army training. As they were all new inductees, none had ever before fired army weapons of any description. Two distinctly separate studies were carried out.

EXPERIMENTAL STUDY 1

Method.—The men tested were for the most part patients who presented themselves to the eye, ear, nose and throat clinic at the station hospital for routine examination of their vision. A few had diseases of the nose and throat, but only those were tested whose complaints would in no way affect the results of the study.

Each subject was questioned relative to deafness in his family and was asked whether he had ever fired a gun or worked in a noisy place, whether he had at any time taken quinine and whether he had ever noted tinnitus or had otitis media. The ears of each subject were examined by means of an ear speculum and a head mirror.

TABLE 2.—*Data on the Series of One Hundred and Eight Subjects in Study I*

Subject *	Family History of Deafness †	History of Otitis Media ‡	Scarred Membrana Tympani §	Coached	Cotton in Ears While Firing ‖	Subjective Deafness After Firing#	Tinnitus After Firing #	Ingestion of Quinine
1	..	..	++++	..	+	..	+	+
2	..	..	..	..	..	+R	+	..
3	..	+R	+	..	..	..	..	..
4	..	..	+	..	+	+	++L	..
5	..	+L	+	..	..	..	+	..
6	F	..	..	..	+	+	++	+
7	..	..	..	..	..	..	+	..
8	..	..	..	..	½+	+	+	..
9	F	..	..	..	..	..	++	..
10	..	..	..	..	..	..	..	..
11	..	..	..	..	..	..	+	..
12	..	..	..	..	..	..	+	..
13	..	+L	++	..	..	..	++	+
14	..	+	..	+	..	..	+	..
15	..	..	..	..	..	..	..	..
16	..	..	..	+	..	..	..	..
17	..	..	++	+	½+	+	+	..
18	..	..	..	..	..	..	+	..
19	..	..	+R	..	..	..	+	..
20	..	..	..	..	..	..	..	..
21	..	..	..	..	..	..	+	..
22	..	..	..	..	½+	..	++	..
23	..	..	..	..	..	..	+R	..
24	..	..	++	+	..	+	+	..
25	F	..	+	..	..	+	..	..
26	..	..	..	..	..	..	++L	..
27	..	..	..	..	..	..	+	..
28	..	..	..	..	..	..	..	..
29	..	..	++	..	..	..	++	..
30	..	..	+L	+	..	+	..	..
31	M	..	..	..	..	..	..	..
32	..	..	..	..	..	..	+	..
33	..	..	..	..	..	..	..	..
34	..	..	..	..	..	+	+	..
35	..	..	..	..	..	..	++L	..
36	..	..	..	..	..	..	++L	..
37	..	..	..	..	..	..	++L	..
38	F	..	+	..	..	..	++L	..
39	..	..	..	..	..	..	++L	..
40	..	..	..	..	..	+	+	..
41	..	..	..	+	..	+	..	+
42	..	..	..	+	..	..	..	..
43	..	..	..	..	½+	+	+	..
44	..	..	..	..	..	..	+	+
45	..	..	..	..	+	..	..	..
46	..	..	..	..	..	..	..	..
47	..	..	..	+	+	..	+	..
48	..	..	..	..	..	..	..	..
49	..	..	..	..	..	..	+	+
50	..	..	..	+	½+	..	..	+
51	..	..	+	..	..	..	..	..
52	..	+	+++L	..	..	..	..	+
53	..	..	..	+	..	..	..	..
54	..	..	..	..	..	..	..	..
55	..	..	++	+	..	..	+	..
56	..	..	++	..	..	..	+	..
57	..	..	+	..	..	..	..	+
58	..	..	..	..	..	..	..	..
59	..	..	..	..	..	..	..	..
60	..	..	++L	+	..	..	++	..
61	..	..	..	..	½+	..	..	..
62	..	..	..	..	..	..	..	..
63	..	..	..	..	..	..	..	..
64	..	..	..	..	..	..	..	..
65	..	..	..	+	+	..	+L	..
66	..	..	..	..	..	..	..	..
67	..	..	..	..	..	..	..	..
68	..	..	..	..	..	..	..	..
69	..	..	..	..	..	..	..	..
70	..	..	..	..	..	..	..	..
71	..	..	..	..	+	..	..	..
72	..	..	..	..	+	..	..	..
73	..	..	..	..	..	..	..	..
74	..	+	++	+	..	+	+	..
75	..	..	..	..	..	..	..	..
76	..	..	..	..	..	..	..	..
77	..	..	..	..	+	..	..	..
78	..	..	..	..	..	..	..	..
79	..	..	..	..	..	..	..	..
80	..	..	..	..	..	..	..	..
81	..	..	..	..	..	..	+	..
82	..	..	..	..	+	..	..	..
83	F	..	..	+	..	..	..	..
84	..	..	+	..	½+	..	+	..
85	..	..	..	..	..	..	+L	..
86	..	..	..	..	..	..	..	..
87	..	..	..	..	..	..	..	..
88	..	..	..	..	..	..	..	..
89	..	..	+	..	..	..	++L	..
90	..	..	..	..	..	..	..	..
91	..	..	..	..	..	..	..	..
92	..	+R	..	..	..	..	..	..
93	..	..	..	..	+	..	+	..
94	..	..	+	..	..	..	+	..
95	..	..	..	..	..	..	..	..
96	..	..	++	..	..	..	..	..
97	..	..	+++R	..	+	..	..	..
98	..	..	..	+	+	..	..	+
99	..	+R	+++	+	+	..	..	..
100	..	..	+L	..	..	..	..	..
101	..	+	..	+	+	..	..	..
102	..	..	..	+	+	..	..	..
103	..	..	..	+	+	..	..	..
104	..	+	..	+	..	..	..	..
105	..	..	..	..	..	..	+	..
106	..	..	..	..	..	..	+	..
107	..	+	..	+	+	..	+	+
108	..	..	++	..	..	..	..	..
Totals	6	11	27	22	25	13	48	11

* Subjects have been renumbered to facilitate grouping.
† F indicates father, and M, mother.
‡ R indicates the right ear, and L, the left ear.
§ The number of pluses indicate the degree of scarring on the basis of + to ++++. When it is not otherwise designated, scarring is bilateral.
‖ ½+ means part time.
+ means subjective complaint of deafness or tinnitus after firing; ++, subjective complaint still present next morning.

4. Wilson, W. H.: Prevention of Traumatic Deafness: A Preliminary Report, Arch. Otolaryng. **37**:757-767 (June) 1943.

A complete audiogram was obtained for each ear. The right ear was then exposed to a fatiguing tone of 2048 cycles per second at 80 decibels intensity for eight minutes. After this, one minute was allowed to elapse, and then a complete audiogram was taken for the exposed ear. The left ear was fatigued and tested in the same manner.

All fatiguing tests and audiograms were made with a Western Electric no. 6a audiometer. Readings were recorded at 5 decibel intervals. A soundproof room was not available, but a relatively quiet room in the hospital was set aside for the testing.

All the men tested were instructed to report back the morning after they completed their basic firing on both the pistol and the rifle range. In no instance were they known to have gone out on the range sooner than five days after being exposed to the fatiguing tone.

The course of firing consisted of 12 rounds with a .45 caliber pistol and 70 rounds with a .30 caliber rifle, of which 40 rounds were rapid fire and 30 rounds slow fire. In addition to the acoustic trauma of the firing of their own weapons, they were subjected to that of the firing of other arms in close proximity. Twenty-two men of the series also coached. A firing coach stands to the right and just in front of his pupil. He must face the firer in order to perform his duty properly, and in this position he is more exposed to the muzzle blast of the gun than if he were actually firing. This is particularly true of his right ear. The subjects spent from two to three days on the firing range with the exception of 2 who completed their course in one day.

Between the firing and the next audiometric study there was a minimum lapse of thirteen hours and a maximum lapse of twenty-five hours except that one subject returned five days later.

On their return the subjects were asked whether they used ear plugs of any sort, whether they coached as well as fired and whether they noted deafness or tinnitus after firing.

Complete audiograms were again made on all subjects as they returned. Those who showed an increase over the originally determined threshold of 10 decibels or more for one or more tested frequencies were instructed to return in seven days.

Observations.—The average age of the 108 subjects was 25.0 years. Six soldiers gave a history of deafness in the family. In each instance, however, but one member was affected. Three of these 6 soldiers showed auditory fatigue and loss after firing. Two showed loss after firing but not fatigue. Only 1 with a family history of deafness failed to show either fatigue or a rise in threshold after firing. In that not more than one member was reported as being deaf in any one family it cannot be stated that the affliction was in any case definitely familial.

None of the men had at any time fired an army rifle. Two of the series had previously worked in a canning factory. Two had worked in a steel mill. Two had been pneumatic hammer operators. One had worked in a shipyard. Two had run a tractor at some time, and one of these had also worked in a wire mill. Thirty-three of the men had previously hunted with a shotgun or fired a .22 caliber rifle. Of these, 2 had also run a tractor, 2 had worked in a steel mill, 1 had been a riveter and 1 a blacksmith. The responses of these men to the fatiguing tests and the trauma of firing did not differ appreciably from the responses of the whole series. Their initial average threshold curve was also almost identical with that of the entire group.

Six men had experienced previous attacks of tinnitus. All of them showed evidence in their audiograms of loss for high tones. One also had conduction deafness. This man and 1 other had previously done a moderate amount of hunting. Four of the 6 men showed definite fatigue; the same 4 subjects had a rise in threshold when they returned from the firing range.

Eleven men gave a history of having had one or more attacks of acute otitis media. Twenty-seven subjects presented some degree of scarring of one or both tympanic membranes. As a group, however, these men gave about the same responses in the tests as did the others.

As in a previous series [4] the minimal loss in decibels which would be called a deviation from the normal was established, the normal for each subject being his original threshold curve. A deviation of less than 10 decibels was again considered to be within experimental error. Any repeat audiogram showing a rise in threshold of 10 decibels or more above the original level for one or more of the tested frequencies was classified as indicating a loss. Of the total series, 30 soldiers fell in this category after being subjected to the fatigue tone. The remaining 78 were classified as not showing fatigue. This group of 30 men were expected to have a definite loss of hearing on their return from the firing range at a later date, and 25 did show such a loss according to the standard. Five of the group after firing gave the same threshold curve that they had given before.

Of the 78 men who did not show fatigue, 14 had a rise in threshold on returning from the firing range. Sixty-four demonstrated no change.

The men who showed a hearing loss after firing were instructed to return seven days later. Of the 39 subjects who were instructed to return, only 26 did, since many of the men were sent to other posts shortly after the completion of their basic training.

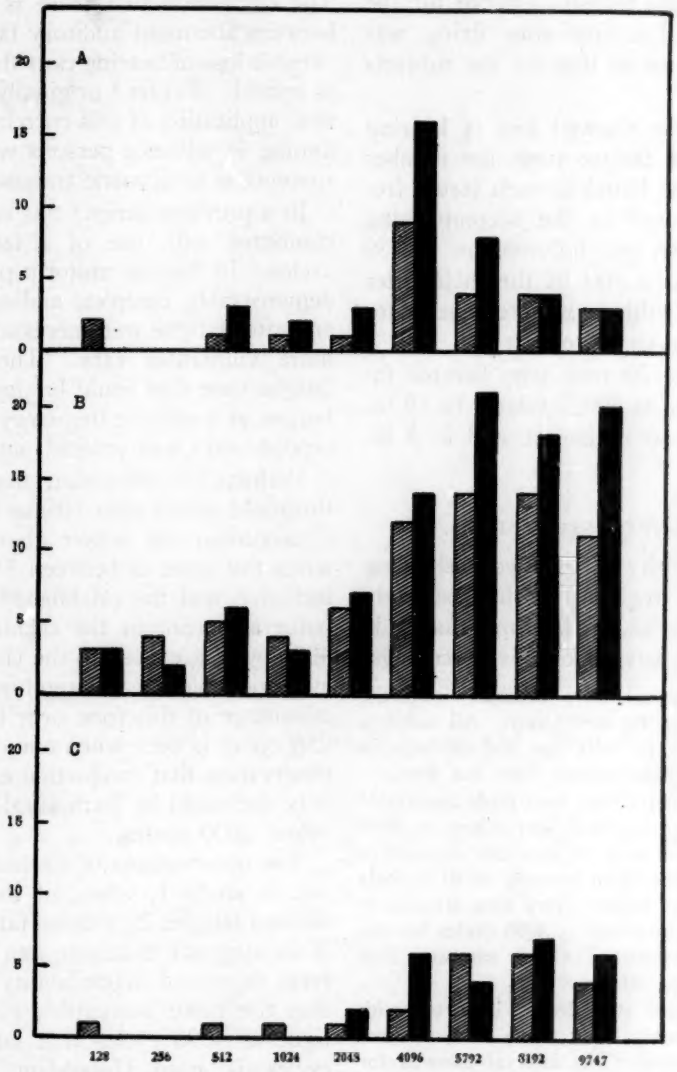
Sixteen of these 26 subjects still showed some loss at the end of the seven day period. Five of the 10 subjects who regained the hearing lost were in the group for whom it was originally predicted that their threshold curves would remain stable. In this group only 1 soldier who returned

after the week's interval retained the loss of hearing from firing. In 5 cases a minimal rise in threshold was seen at frequencies at which previously such a rise had not been demonstrated. One of these subjects was suffering from an acute infection of the upper respiratory tract at the time the final threshold curve was determined.

Of the 39 subjects showing after-firing loss, 11 complained of deafness after firing. None of these were aware of any deafness when audiograms were taken the next day. Of the 69 subjects not showing after-firing loss, 2 complained of deafness after firing. None complained the next day.

Of the 39 subjects showing after-firing loss, 29 complained of tinnitus after firing. Ten of these still complained of tinnitus the next day. Of the 69 subjects not showing after-firing loss, 19 complained of tinnitus after firing. Four still complained of tinnitus the next day.

Twenty-five of the men wore plugs in their ears while firing. These were made of either cotton or rifle-cleaning patches. Seven of these 25 men wore them only part time. The percentage of rises in threshold after firing was approximately the same for this group as for the entire series.



Number of subjects in study I in whom a rise in threshold was observed at each frequency tested: (*A*) after fatiguing, 30 subjects; (*B*) after firing, 39 subjects, and (*C*) after one week's recovery, 26 subjects. Solid black indicates values for the left ear; cross hatching, values for the right ear.

Eleven men gave a history of having taken appreciable amounts of quinine at some time in the past. The responses of these men to the fatiguing tests and the trauma of firing were practically the same as the responses of the entire series. There was an interesting difference in their average original threshold curve as compared with the average for those who

gave no history of having ingested quinine. There was no appreciable difference in the two average curves for the first five frequencies tested. At 4096 cycles the average curve for the 11 right ears was 13.0 decibels higher and that for the left ears was 14.8 decibels higher. At 5792 cycles the curves were 14.1 decibels and 16.4 decibels higher respectively, at 8192 cycles, 11.5 decibels and 13.6 decibels higher, and at 9747 cycles, 7.1 decibels and 14.7 decibels higher.

The average original threshold curve for the subjects who showed a rise after firing was approximately the same as that for the subjects who did not.

Of the 30 men who showed loss of hearing after exposure to the fatigue tone, the number for whom fatigue was found at each tested frequency is demonstrated in the accompanying chart. The same plan was followed for the 39 subjects who showed a rise in threshold after firing and for the 26 subjects who returned later for checks on the maximal recovery.

Twenty-two of the 30 men who became fatigued showed fatigue at 4096 cycles. In 19 instances the fatigue was unilateral, and in 3 instances, bilateral.

EXPERIMENTAL STUDY II

In that the majority of subjects showing fatigue at any tested frequency in the first study showed it at 4096 cycles, an attempt was made to determine the consistency of this percentage.

Method.—The men tested were obtained from the same source as those in the first study. All subjects were questioned relative to their age and deafness in their family. Physical examinations were not done.

After the individual subject had been made acquainted with the audiometer, a threshold was taken at 4096 cycles for each ear. The right ear was then exposed to the 2048 cycle fatigue tone at an intensity of 80 decibels for eight minutes. Two minutes were then allowed to elapse, after which the threshold at 4096 cycles for the right ear was again taken. The left ear was then fatigued and tested in the same manner.

Readings were recorded at 5 decibel intervals, with a loss of 10 decibels again being taken as a minimal deviation from the normal. The interval allowed for recovery before redetermining the threshold was changed to two minutes to approximate more closely the average interval when all frequencies are tested.

Observations.—Of the 268 subjects exposed to the fatigue tone, 66 showed fatigue. Twenty-three of these fatigued bilaterally. Seventeen showed fatigue in the right ear only, and 26 in the left ear only.

The ratio of these 66 cases to the 268 closely approximates the ratio of the 22 cases showing fatigue at 4096 cycles to the 108 total in study I, the ratio being 1 to 4.1, compared with 1 to 4.9, respectively.

The average age for the 268 subjects was 26.1 years. Twenty-one men gave a history of deafness in the family. In no instance was more than one member affected. Seven of the 21 men showed fatigue.

COMMENT

These experimental studies demonstrate the greater susceptibility of some ears over others. The contention that there is a definite relation between abnormal auditory fatigue and the irreversible loss of hearing caused by acoustic trauma is upheld. Peyser [5] originally suggested a practical application of this correlation, that of determining in advance persons who may be unduly susceptible to acoustic trauma.

In a previous series [4] this correlation was substantiated with use of a fatigue tone of 256 cycles. In that no uniform point of fatigue was demonstrable, complete audiograms made before and after fatigue were necessary to determine the more vulnerable ears. The desirability of a fatigue tone that could be depended on to cause fatigue at a definite frequency in these more susceptible ears was pointed out.

Perlman's [6] conclusion that when a rise in threshold occurs after fatigue it generally reaches a maximum one octave above the fatigue tone when the latter is between 512 and 4096 cycles inclusive, and the established fact that the most vulnerable spot in the cochlea is found in the 4096 cycle area, led to the choice of 2048 cycles as a possible ideal fatiguing tone. A further advantage of this tone over the previously used 256 cycles is seen when one considers Wiggers' [7] observation that conduction efficiency is appreciably decreased by intra-aural muscle action only below 1000 cycles.

The observations of Perlman were well borne out in study I, when, of the 30 subjects who showed fatigue, 22 showed fatigue at 4096 cycles. This suggests that one can count on a moderate degree of dependability in the observation that the more susceptible ears will present fatigue at 4096 cycles if a fatigue tone of 2048 cycles is used. Upholding this dependability further is the fact that the ratio of those that showed fatigue at 4096 cycles to the entire series in study II is very close to the ratio of the same respective groups in study I.

5. Peyser, A.: Zur Methodik einer otologischen Prophylaxis der industriellen Larmschwerhörigkeit, Acta otolaryng. **28**:443-462, 1940.

6. Perlman, H. B.: Acoustic Trauma in Man, Arch. Otolaryng. **34**:429-452 (Sept.) 1941.

7. Wiggers, H. C.: Functions of the Intra-Aural Muscles, Am. J. Physiol. **120**:771-780 (Dec.) 1937.

TABLE 3.—*Rise Above Original Threshold Curve in Study I, Expressed in Decibels (a) After Fatiguing, (b) After Firing and (c) After Recovery for One Week* *

Subject	128 R	128 L	256 R	256 L	512 R	512 L	1024 R	1024 L	2048 R	2048 L	4096 R	4096 L	5792 R	5792 L	8192 R	8192 L	9747 R	9747 L
								Frequencies										
								Threshold Curves Correctly Predicted to Rise										
1 (a)	..	..	..	..	..	10	..	..	..	10	20	..	..	..	..	..	..	..
(b)	10	10	10	10	10	15	..	..	..	..	..	..	..	..	..	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
2 (a)	..	..	..	..	..	..	..	..	..	10	..	20	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	20	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
3 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	10
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..
4 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	15	..	..	15	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
5 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
(c)	Did not return																	
6 (a)	..	..	..	..	..	..	..	..	..	..	..	15	..	..	10	..	..	..
(b)	..	..	..	..	..	..	..	..	..	10	..	10	..	..	10	25	..	..
(c)	..	..	..	10	..	..	..	10	..	10	10	15	10	10	15	30	..	..
7 (a)	..	..	..	..	..	..	..	..	..	..	..	15	10	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	15	10	..	..	10	20	10
(c)	..	..	..	..	..	..	..	..	..	..	..	15	10	..	..	10	20	..
8 (a)	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	20	..	10	25	..	25	20	45
(c)	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..
9 (a)	..	..	..	..	..	..	..	..	..	..	10	10	10	15	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	30	45	10	20	15	10	..	20
(c)	..	..	..	..	..	..	..	..	..	10	..	20	20	20	15	10	..	15
10 (a)	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	45	..	35	20	30	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	35	..	20	10	20	..	..	..
11 (a)	..	..	..	..	..	..	..	..	..	..	..	35	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	25	..	10	..	..	..	10
(c)	..	..	..	..	..	..	..	..	..	..	..	25	..	..	..	..	..	10
12 (a)	..	..	..	..	..	15	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	10	..	..	..	..	..	..	..	..	..	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
13 (a)	..	..	..	..	..	..	..	..	10	20	10	15	..	20	15	..	20	15
(b)	..	..	..	..	..	..	..	..	10	..	20	..	15	15	20	10	30	10
(c)†	..	..	..	..	..	..	..	..	..	15	15	30	40	25	35	25	50	45
14 (a)	..	..	..	..	..	..	..	..	..	..	..	15	..	15	..	10	..	..
(b)	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..	..	..	..
(c)	Did not return																	
15 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	15	..	15	..	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
16 (a)	..	..	..	..	..	..	..	..	..	..	..	..	10	..	15	25	..	..
(b)	..	..	..	..	..	10	10	15	20	20	25	10	20	..	25	25	20	20
(c)	Did not return																	
17 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
(b)†	..	..	10	..	10	..	10	..	10	..	25	20	20	20	25	20	..	..
(c)	..	..	..	..	..	..	10	..	10	..	..	..	..	..	25	..	..	..
18 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	30	40	..	20	..	40
(c)	Did not return																	
19 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
(b)	15	30	15	10	10	15	10	10	20	15	15	50	25	30	15	20	25	40
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
20 (a)	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..	..	..
(b)	..	..	..	..	15	..	10	..	10	..	20	15	25	..	..	..	..	..
(c)	..	..	..	..	10	..	..	..	..	..	..	..	10	..	..	..	..	..
21 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..
(c)	Did not return																	
22 (a)	20	..	..	..	10	..	15	..	..	..	10	..	..	..	..	10	..	..
(b)	20	10	..	..	..	15	..	..	..	..	20	25	..	..	..	..	15	15
(c)	10	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	45
23 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..	..
24 (a)	..	..	..	..	..	10	..	10	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	10	..	..	..	20	35	30	..	20	20	20
(c)	..	..	..	..	..	..	..	..	..	10	..	10	..	..	..	..	10	..
25 (a)	10	..	..	..	..	..	..	..	..	..	10	..	..	..	15	..	..	10
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..	10
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
								Threshold Curves Predicted to Rise That Remained Stable										
26 (a)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	10	10
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(c)	Not asked to return																	
27 (a)	..	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(c)	Not asked to return																	

* R indicates the right ear; L, the left ear.
† An acute infection of the upper respiratory tract was present at the time the threshold curve was taken.

TABLE 3.—*Rise Above Original Threshold Curve in Study I, Expressed in Decibels (a) After Fatiguing, (b) After Firing and (c) After Recovery for One Week*—Continued*

Subject	Frequencies																	
	128		256		512		1024		2048		4096		5792		8192		9747	
	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
Threshold Curves Predicted to Rise That Remained Stable																		
28 (a)	..	..	..	..	..	..	..	..	..	..	15	15	10	10	..	..	10	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(c)	Not asked to return																	
29 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(c)	Not asked to return																	
30 (a)	..	..	..	..	..	..	..	10	..	..	..	10	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(c)	Not asked to return																	
Threshold Curves Predicted to Remain Stable That Rose																		
31 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..
(c)	Did not return																	
32 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	15	35	..	..	..	..
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
33 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..	..
(c)	Did not return																	
34 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	10	20	10	..	15	15	15	..	15	25
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
35 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	15	..	25
(c)	Did not return																	
36 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	50	..	30	..	30	..	35	..	50
(c)	Did not return																	
37 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	15	..	..	15	..	..	..	..
(c)	Did not return																	
38 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	10	..	..	..	30	..	50	..	60	..	60	..	60
(c)	Did not return																	
39 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	25	..	10
(c)	Did not return																	
40 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	35	..	10	..	15
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
41 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	45	20	35	10	45
(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	40	30	30	15	10
42 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	15	..
(c)	Did not return																	
43 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)†	..	..	..	..	..	..	..	..	..	..	35	..	25	15	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
44 (a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	10	..	10	..	..	..	..	..	..	..	..	..	..	..	..	..
(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Threshold Curves Correctly Predicted to Remain Stable																		
45 to 108	No change in original threshold curve after exposure to fatiguing tone or after firing																	

The percentage of threshold curves correctly predicted to rise in study I with use of 2048 cycles as a fatigue tone closely approximates the percentage correctly predicted in a previous series [4] with use of 256 cycles as a fatigue tone. It cannot be said that a higher percentage of susceptible ears were discovered by using a tone of 2048 cycles as the fatigue tone, but the relative consistency of fatigue one octave above this frequency is encouraging from the standpoint of simplifying the method of screening. The determination of the hearing threshold at 4096 cycles of the person applying for work in a noisy industry, exposure of the candidate to a fatigue tone of 2048 cycles, and then redetermination of the threshold should be a relatively short procedure. If a man is refused work in a noisy industry because his ears are susceptible to acoustic trauma, not only has he benefited, but so too has the industry that would have employed him.

With compensation commissions turning more and more toward compensating for industrial losses of hearing, the prevention of these losses looms ever larger as the one means of meeting this trend. With industry becoming more rather than less noisy, the one hope lies in the screening out in advance of persons predisposed to traumatic deafness.

SUMMARY AND CONCLUSIONS

The trend is toward allowing compensation for traumatic impairment of hearing.

The ears of persons showing abnormal auditory fatigue are more predisposed to irreversible traumatic loss of hearing.

The realization of such a relation affords a means of screening out susceptible persons as they apply for work in noisy industries.

A large percentage of persons whose ears fatigue abnormally will show auditory fatigue at 4096 cycles if exposed to a fatigue tone of 2048 cycles.

The discovery of a moderately dependable tonal frequency to show abnormal fatigue suggests a relatively simple procedure for determining in advance persons predisposed to traumatic deafness.

Clinical Notes; New Instruments and Technics

MODIFICATION OF THE BLADE OF THE LaFORCE TONSILLECTOME

FREDERICK E. KELLER, M.D., PHILADELPHIA

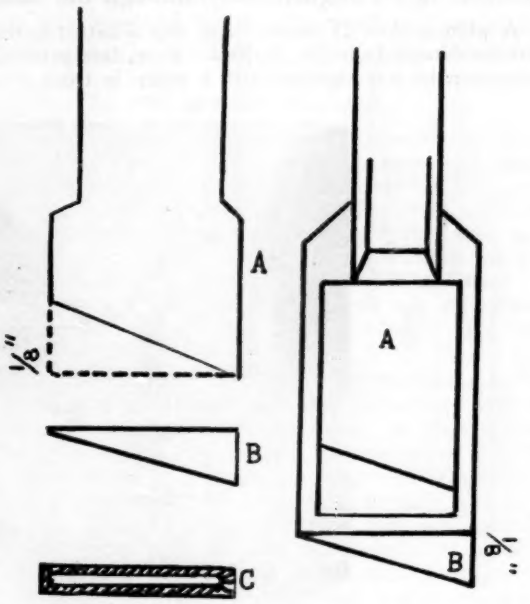
Those who use the LaForce tonsillectome have noted that when the blade gets dull there is difficulty in disengaging an incompletely severed tonsil because the tissues have not been completely cut through with the blade.

The modification of this blade presented here is based on the principle that cutting is usually not done by cutting instruments straight across but rather at an oblique angle, e. g., cleavers, the guillotine or the angle of a cutting saw. The blade has been modified for such a purpose. It has been made longer, ⅛ inch (0.32 cm.) on one side, so that cutting begins at first with the longer side until the tonsil has been completely severed.

To prevent damage to the tissues, an additional guide had to be added to the frame to accommodate the longer end of the blade. This guide is welded to the bottom of the blade carrier and is shaped the same as the cutting edge of the blade about ⅛ inch on the longer side and 1/16 inch (0.08 cm.) on the shorter side. The instrument has now been used in many cases and found to be excellent. So far no tonsil has been incompletely severed, even when accidentally the middle of the tonsil had been engaged. Any instrument maker could modify the old La Force tonsillectome by making a new blade and welding a guide to the old frame. In Philadelphia, Brotherston Surgical Co., 2214 Chestnut Street, have modified this one.

Anderson Hospital, 1711 Green Street.

Presented at the joint meeting of the Philadelphia Laryngological Society and the Section on Otolaryngology of the College of Physicians, of Philadelphia, Oct. 20, 1943.



Modified blade and guide for the LaForce tonsillectome: *A*, new blade; *B*, guide welded to old frame; *C*, slot in guide to permit blade to enter safely.

Case Reports

AN UNUSUAL FOREIGN BODY OF THE LARYNX

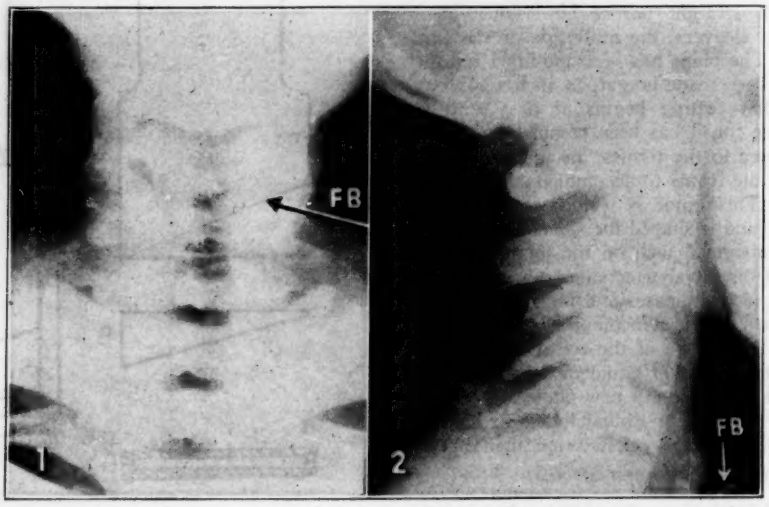
MAJOR ARNOLD F. JUDGE*
MEDICAL CORPS, ARMY OF THE UNITED STATES

A foreign body of the air passages which enters by an avenue other than the glottis is rare. Cases are of sufficient interest to the laryngologist to warrant their publication. In discussing foreign bodies of the air passages from the standpoint of etiology Jackson [1] stated: "A penetrating projectile may be inspirated. We have had one such case."

In the case to be reported now, a metallic foreign body pierced the skin of the neck and the cricothyroid membrane and came to lodge in the ventricle of the larynx on the side opposite to the side of its original entry through the skin.

A white soldier, 21 years of age, was admitted to the Station Hospital on Jan. 5, 1944. Four days prior to admission he was standing with a group in front of a campfire when one of the group threw a .32 caliber blank cartridge into the fire. An explosion followed, and the patient was struck in the neck by one of the shell fragments. A penetrating wound of the neck resulted. The patient was seen at the Battalion Aid Station, where tetanus prophylaxis was administered and the wound closed with one silk suture. The patient was then sent to the Evacuation Hospital because of loss of voice and hemoptysis. Four days later he entered the Station Hospital. A roentgenologic report accompanied the patient. This report stated: "There is an opaque foreign body 1 cm. long and 2 mm. wide in the left side of the neck at the level of the false vocal cords at the midline. One end of this foreign body appears lodged in the left side of the larynx."

Examination in the Eye, Ear, Nose and Throat Clinic on January 6 revealed a wound in the right side of the neck at the level of the upper margin of the cricoid cartilage and just to the right of the midline. The wound had healed by first intention, so a black silk suture was removed. No foreign body could be felt subcutaneously on either side of the neck. The left side of the nose was filled with thick mucoid exudate, and the same exudate was present in the posterior part of the pharynx. Mirror examination of the larynx revealed swelling of both false cords and some crusting exudate in the posterior commissure. What was either a small, flattened piece of metal or crusting exudate was seen in the left ventricle near the anterior commissure.



Anteroposterior (1) and lateral (2) views of the neck showing an opaque foreign body in the ventricle of the larynx on the left.

Another roentgenogram of the neck was made to see whether the foreign body described at the Evacuation Hospital was still in place. In the meantime, measures were taken to clear up the discharge from the nose and the sinuses. On January 11 the larynx was again examined with a mirror. The swelling of the false cords had subsided, and a metallic foreign body could be seen protruding about 2 mm. into the glottis. The roentgenogram revealed that the foreign body was still situated as described previously.

On January 13 direct laryngoscopy was performed, and a small metallic foreign body was disengaged with little difficulty from the tissues of the left laryngeal ventricle and removed with an alligator forceps.

The patient was discharged five days later with no residual symptoms. At this time a fibrous tract could be palpated leading from the scar on the right side of the neck, upward and medially to the cricothyroid membrane.

*Instructor in otolaryngology at Albany Medical College, on leave of absence.

1. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

ADAMANTINOMA OF THE MAXILLARY SINUS

WALTER F. MOSHER, M.D., VENTURA, CALIF.

This type of tumor is called adamantinoma in most textbooks; however, it appears under several other classifications, such as ameloblastoma or adamantoblastoma. The first complete description is usually credited to Folkson (1879). Since then many others have reported cases, but each one pointed out the rarity of the growth, and no one until recently has surveyed the previous reports.

Robinson [1] and Thoma,[2] who reviewed the literature and probably have written more extensively on this type of tumor from the point of view of pathology than any one else stated that it may be defined as an epithelial tumor, arising from the odontogenic apparatus or from cells with a potentiality of forming tissues of the enamel organ. It never contains enamel and is either solid or cystic. The odontoma, which is the other tumor arising from the odontogenic cells, is made up of two or more tissues and is most often a hard calcified structure.

While the adamantinoma is found most frequently in the mandible or in the maxilla, it is also occasionally seen in other parts of the body, e. g., the pituitary gland and the tibia. In over 80 per cent of the cases it occurred in the mandible, and in about 15 per cent in the maxilla. It occurs about equally in males and females, the average age of patients at the time it is first recognized being a little past 30. The tumor apparently remains quiescent for years and then, due to some irritation, such as an injury, a dental procedure or an infection, it takes on more active growth. When the tumor develops in the maxilla, the maxillary sinus eventually becomes involved, and the tumor then comes within the scope of the rhinologist.

In the maxilla the growth is usually of a solid glandular character, and shortly shows signs of gradual growth, eventually reaching considerable proportions. History reveals that in few cases does it metastasize, and it is not considered to be a truly malignant tumor.

Robinson has described this tumor as nonfunctional, intermittent in growth, anatomically benign but clinically persistent. Like all other such tumors, it may take on malignant characteristics and is then usually classified under the term adamantocarcinoma or adamantosarcoma. For the removal of the growth a rather radical procedure is usually recommended; New recommended its removal with electrodiathermy. If all the tumor is not removed, there is a great tendency for it to recur. Likewise it is thought to be quite resistant to both roentgen and radium therapy, although a few cases have been reported in which cures were effected by the use of radiation therapy, following nonremoval of the growth. While the adamantinoma is not rare, Robinson in 1937 having compiled a list of 379 cases from the literature, yet the number of cases in which it has involved the maxillary sinus is small and warrants the description of a typical instance.

REPORT OF A CASE

R. R., a white American aged 61, presented himself at my office Dec. 26, 1941, relating a history of having been struck over the left maxilla by the lid of an automobile trunk while placing ice in the trunk. This injury occurred about three months before he sought advice as a patient. He complained of moderate pain over the maxilla, as well as of considerable nasal obstruction and discharge from the left side of the nose, with some swelling over the left maxilla. He complained of no other symptoms.

On examination it was found that the lateral wall of the nose on the left side had been pushed over until the turbinates touched the septum. Some thin discharge and a slight swelling over the maxilla were noted. The left side of the hard palate was also depressed. Transillumination showed the left antrum to be dark, the other sinuses all being clear. Believing that this condition might be due to injury and that there might be some infection with necrotic bone in the antrum, I sent the patient out for roentgen study, after first reporting the matter to the insurance company, since the patient was insured. The blood and the urine were normal. The Wassermann test was negative. The roentgen examination revealed the antrum to be filled with a large mass and the anterior bony wall of the maxilla to be eroded. The appearance was that of a new growth. Permission to operate on and explore the antrum was received from the insurance company.

With the patient under anesthesia induced with pentothal sodium, the antrum was opened by the Caldwell-Luc method, and the entire cavity was seen to be filled with a large, rather spongy mass. The anterior wall was eroded, and the remaining bone had been thinned out. I removed as much of the tumor as possible, but the bleeding was so severe that after taking out about one third of the mass, I had to stop and pack the antrum.

The specimen was then sent to the laboratory of Zeiler, Hammack and Maner for examination, and the following report was received:

"The specimen consists of numerous small irregular pieces of reddish gray fixed tissue. Microscopic exami-

---

Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, Jan. 23, 1944.

1. Robinson, H. B. G.: Ameloblastoma: A Survey of Three Hundred and Seventy-Nine Cases from the Literature, Arch. Path. **23**:831 (June) 1937.

2. Thoma, K. H.: Oral Pathology, St. Louis, C. V. Mosby Company, 1941.

nation of this tissue shows it to be made up of epithelial cells of the enamel type, arranged in long columns and alveoli separated by fibrous connective tissue. Many imperfect alveolar spaces are seen filled with eosin-staining structureless material and lined with several layers of epithelial enameloblasts. Scattered throughout the stroma are numerous dilated vascular spaces filled with red blood cells. In one area the tumor is degenerated and necrotic. The histologic appearance is typically that of glandular adamantinoma.

"Diagnosis: Glandular adamantinoma.

"Note: Since the epithelial elements in this tumor are so well differentiated, we expect it to be radioresistant. The tumor is slow growing, and its natural history is prolonged. It therefore cannot be regarded as the direct result of recent injury."

The patient indicated his preference for a conservative surgical procedure. Because of this and because of his age, it was concluded that a radical resection should not be done. On Jan. 17, 1942, nine days after the first operation, the antrum was reopened with the patient under pentothal sodium anesthesia. Because of the excessive bleeding encountered before, the external carotid artery was tied, and this time it was possible to proceed in a comparatively dry field. Using sharp dissection and curettage I removed what I thought was all of the growth. The mass was about 3 by 3 by 5 cm., so that when the tumor had been dissected out the cavity was about twice the size of a normal antrum.

Following the removal, the patient was given rather intensive roentgen therapy for approximately two months. Shortly after this the incision closed with no fistula or other untoward incident, and two years have now passed with no apparent recurrence of the tumor. A roentgen examination made just a short time prior to writing revealed no evidence of recurrence.

Examination of the patient at the time of writing shows about the same amount of passageway in each nostril, and the patient is able to breathe through both sides of the nose, which he could not do previously. However, on transillumination the left antrum is still dark, probably because of the fibrosis following the operation.

COMMENT

When the report came back from the pathologist, a more explicit history was taken and it was learned that about five or six years prior to the injury the patient had had a third molar tooth removed from the left maxilla. At that time the dentist curetted what he told the patient was a small cyst at the base of the molar. It was presumed that the entire mass had been removed, and no pathologic examination of the specimen was made. It is reasonable to suppose that part of the cyst wall was not removed and that this irritation was the inception of the subsequent tumor growth.

34 North Ash Street.

Abstracts from Current Literature

Larynx

CHONDROMA OF THE LARYNX: REVIEW OF THE LITERATURE AND REPORT OF TWO CASES. JULIUS W. MCCALL, S. M. DUPERTUIS and FREDERICK S. GARDINER, Laryngoscope **54**:1 (Jan.) 1944.

The authors present 2 interesting cases of chondroma of the larynx. They review the literature on the subject, noting that there is a total of but 85 cases reported.

The sites of these lesions in order of frequency are the cricoid cartilage, the thyroid cartilage, the epiglottis and the arytenoid cartilage. The growth usually extends into the larynx, obstructing the airway. It is usually described as round, solitary and immobile; it is covered with normal mucous membrane. The movement of the cords may be interfered with. The growth may be very small or so large as to obstruct the larynx completely.

The histologic appearance of the tumor resembles that of the cartilage from which it originates, but with some modifications, which the authors describe. The tumors do not metastasize, but they do tend to recur after removal.

The majority of the patients are between the ages of 40 and 60 when the tumor occurs, and are predominantly males.

Symptoms depend on the size and the site of the tumor. There may be hoarseness, dyspnea, cough or occasional dysphagia. They are apt to be mild for a considerable time and cause the patient concern only when actual obstruction to breathing results.

The diagnosis is sometimes missed because of the rarity of the tumor and the difficulty in obtaining a biopsy. Roentgen examination aids greatly, particularly when there is ossification or calcification of the tumor.

The treatment is always surgical. The type of operation depends on the site and the size of the tumor. It must be kept in mind that if any part of the tumor or of the capsule is left, recurrence will follow. Laryngofissure or laryngectomy is therefore often necessary.

HITSCHLER, Philadelphia.

X-RAY DIAGNOSIS OF BRONCHOGENIC CARCINOMA. NEWTON SCATCHARD, New York State J. Med. **44**: 617 (March 15) 1944.

Scatchard says that bronchogenic carcinoma is the second most frequent cancer in the male! The first is carcinoma of the stomach. In his clinic at the Edward J. Meyer Memorial Hospital, Buffalo, he has had the help of a working laboratory staff and of clinical and surgical staffs, all of whom have contributed to the data given. They see two pulmonary carcinomas to every three gastric ones, and "this lesion [carcinoma of a bronchus] kills 15,000 in the United States yearly." This is approximately one tenth of all deaths from cancers. In making an early diagnosis the roentgenologist has a great responsibility, but he cannot make it alone. Bronchoscopy will help, and also "exploratory thoracotomy," which the author says "is not especially hazardous." If the frozen section is positive for carcinoma and the stage is operable, removal without delay is in order. Procrastination is the arch enemy in these cases.

In conclusion Scatchard says that the roentgen ray can demonstrate early bronchial carcinoma. Cancer must be kept in mind and looked for. Bronchoscopy, biopsy, microscopic study and thoracotomy followed by pneumonectomy may lower the present mortality rate. Roentgen therapy is here as elsewhere merely palliative and should be used only in inoperable cases. Six illustrations of roentgen films show the varying types on which this study is based.

VOORHEES, New York.

ACUTE STREPTOCOCCIC LARYNGITIS. ERNESTO RAFFO, Rev. de otorrinolaring. **2**:3, 1943.

Raffo presents a case of acute septic laryngitis due to Streptococcus. Generalized septicemia developed, with localization in the pleura. Thoracotomy was performed, and the patient made a complete recovery.

The author stresses the importance and the seriousness of this infection. The prognosis should be reserved since complications are common and toxemia may be extremely severe. The therapy is essentially supportive —treatment with sulfonamide compounds, intravenous medication, transfusions and symptomatic treatment of complications when possible.

PERSKY, Philadelphia.

Nose

THE LOCAL USE OF SULFONAMIDES IN NASAL AND SINUS INFECTION. NOAH D. FABRICANT, Am. J. M. Sc. **206**:546 (Oct.) 1943.

In this review Fabricant attempts to evaluate the published results of the local use of sulfonamide compounds in the treatment of nasal and sinus infections. He reminds one that chronic sinusitis is not a specific disease and that success in the treatment of it cannot be expected when it is thus considered. He points out that medicaments used intranasally should be nontoxic, should not interfere with ciliary action and should have a p_H value within that which is normal for the human nose, namely, between 5.5 and 6.5. He quotes Futch, who found that a 5 per cent solution of sulfathiazole sodium sesquihydrate exerted an early destructive effect on the nasal mucous membrane of the rabbit. This solution has a p_H value of approximately 10. He also quotes Fletcher, who reported necrosis of the lining of the maxillary sinus following irrigation of the sinus with a 5 per cent and a 10 per cent solution of sulfathiazole sodium over a period of three weeks. He also thinks there is not sufficient evidence, either clinical or laboratory, to support the widespread intranasal use of a sulfonamide compound in solution. He states: "In attempting to establish the usefulness of local applications of sulfonamides to infection of the nose and sinuses, a majority of writers appear to neglect the entire body of knowledge known as nasal physiology." He quotes Lindsay, who found that the use of a 2.5 per cent solution of sulfadiazine with triethanolamine-technical as a spray did not shorten the course of sinus infection. He also correctly points out that the evidence

supporting the intranasal use of sulfonamide compounds is not convincing and that some of the solutions which have been advocated are distinctly harmful to the nasal mucosa.

DAVISON, Danville, Pa.

DIAGNOSIS, TREATMENT AND END RESULTS IN MALIGNANT TUMORS OF THE NASAL SINUSES. G. ALLEN ROBINSON, New York State J. Med. **44**:713 (April 1) 1944.

Robinson, working at the Manhattan Eye, Ear and Throat Hospital, reports 92 cases of cancer of the nasal sinuses, in which treatment was by radium, roentgen rays and surgical operation. In most cases the lesion was well advanced, and "the diagnosis was rarely made early." Symptoms were pain in the cheek and the orbit, nasal obstruction, bleeding and discharge, bulging of one cheek or of the hard palate, protrusion of the eye and tearing. There may be metastases in the brain, the spinal column, the liver and the lungs. The author stresses careful recording of the history and searching examination of the sinuses, including roentgen study and biopsy. The kind of treatment is determined by the extent of growth, the type of cancer and the "life history." As a rule, radical surgical intervention is followed by treatment with radiation (radium). This is in the "early cases." In the late cases (anaplastic), treatment with roentgen rays or radium precedes surgical intervention; the latter is followed by irradiation of the site. Adequate surgical exposure makes effective use of radiation easier. Of the 92 patients, 19 have survived three years or longer. Of these, 1 died seventeen years later without recurrence; 1 died of heart disease seven years after cure of cancer (free of cancer); 1 died of recurrence fifteen years after; 1 was well for six years, and then cancer developed in the opposite ethmoid sinus, and 15 are living from three to seventeen years after treatment. In discussion Dr. A. W. Dowdy, of Rochester, N. Y., directs attention to infection, pain and necrosis, and he wonders whether these could not have been avoided by better screening. He suggests "1 mm. of platinum as a minimum." But he thinks this 20 per cent arrest is "very good." The article is carefully written and is an honest and encouraging report on results obtained in one of the worst cancer sites in the body.

VOORHEES, New York.

SOME OBSERVATIONS ON THE MAXILLARY SINUS. L. CHESTER MCHENRY, South. M. J. **38**:18 (Jan.) 1943.

According to McHenry, the diagnosis of an acute infection of a maxillary sinus is not difficult. A history of an acute cold in the head followed by the characteristic pain, the presence of a purulent discharge in the middle meatus and the observations on transillumination usually suffice to make the diagnosis. In the case of a chronic infection of the antrum the diagnosis may be much more difficult. Examination of the nose and transillumination of the sinuses may reveal no evidence of infection, and antral irrigation may show no gross evidence of it, but cytologic examination of the antral washings may show large numbers of leukocytes. In such a case roentgenograms of the sinuses made after the instillation of iodized oil may be of great value, and cystlike shadows and localized thickenings are significant. Generalized thickening may or may not be significant.

In the treatment of an acute infection of a maxillary sinus the author recommends rest in bed and the use of sedatives and local astringents, such as neo-synephrine hydrochloride or an ephedrine salt, 0.25 per cent in isotonic solution of sodium chloride. Antiseptic solutions in the form of drops and sprays are not recommended. The sulfonamide compounds may be used in selected cases. Antral irrigations are indicated after the acute stage. McHenry uses the middle meatal approach in more than 90 per cent of his cases. The sinus should not be irrigated more than two or three times per week; irrigations should not be continued over a long period. If satisfactory progress is not being made, the nasoantral window operation is advised. In this operation, the opening is made with a perforator and is enlarged in all directions with suitable punches. The author reports results in a series of 80 patients who were operated on in this manner; 60 were cured, 12 were improved, and in 8 the results were unsatisfactory.

SINGLETON, Dallas, Texas.

Miscellaneous

SULFONAMIDE INHIBITING ACTION OF PROCAINE. OSLER L. PETERSON and MAXWELL FINLAND, Am. J. M. Sc. **207**:166 (Feb.) 1944.

Peterson and Finland report 3 cases in which local infection and a sinus tract developed as the result of lumbar puncture in a patient with purulent meningitis. In each case culture of the infected soft tissue yielded pneumococci of the same type as those present in the spinal fluid. They attribute the infection to the use of procaine hydrochloride as an anesthetic and point out that procaine has an inhibiting effect on the bacteriostatic action of the sulfonamide compounds similar to that of para-aminobenzoic acid. This article also includes the report of a case in which localized infection of the soft tissues developed along the needle track at three points where thoracentesis was performed.

DAVISON, Danville, Pa.

THE TOXICITY OF SULFADIAZINE. NORMAN PLUMMER and CHARLES WHEELER, Am. J. M. Sc. **207**:175 (Feb.) 1944.

The toxic reactions that occurred in 1,357 hospitalized patients who were given sulfadiazine orally or sodium sulfadiazine intravenously, or were given both forms of therapy in combination, are tabulated and analyzed.

One patient died of thrombopenic purpura, which was attributed to sulfadiazine.

Eight per cent of 705 patients who received 6 Gm. of sulfadiazine by mouth daily for at least two days and not more than fourteen days showed evidences of toxicity. The renal reaction was the most frequent single toxic manifestation.

With intravenous injection of sodium sulfadiazine the incidence of the renal reaction was almost doubled, and thereby the total incidence of reaction was raised.

The effect on toxicity of previous use of sulfonamide compounds in therapy was observed in 87 patients. In these there was a slightly increased total incidence of reactions, attributable to a slight increase in the incidence of drug rash, drug fever and leukopenia.

The renal comprised more than half of the toxic reactions following sulfadiazine therapy in the entire series. It is indicated that if this reaction is prevented by a proper intake of fluids together with appropriate alkali therapy, the total incidence of toxic reactions from sulfadiazine can be reduced to approximately 4 per cent.

This study indicates a decided superiority of sulfadiazine over the other commonly employed sulfonamide

compounds on the basis of a low degree of clinical toxicity.

This paper is well worth reading.

DAVISON, Danville, Pa.

CLIMATE AND DISEASE. C. A. MILLS, J. A. M. A. **123**:551 (Oct. 30) 1943.

Mills has distinguished himself for many years by first class observations and studies in climatology. In the present article he takes up the problem of human energetics and the relationship to climatic conditions.

Emphasis is placed on tissue combustion since all the factors of vitality have their functional basis in the energy liberated from such combustion.

The ideal climate is the temperate zone since it is here that man's own internal production of heat is highest and the necessity for rapid dissipation of heat greatest.

Man's susceptibility to infection and his chances for survival are conditioned strongly by the ease with which body heat is lost and on the resulting level of tissue combustion allowed him.

The summer freedom from infection of the respiratory tract is attributable in large part to the lessened storminess of that season and the greater freedom from chilling.

Studies on animals and man have shown that ten days to two weeks are required for any considerable subsidence of basic internal combustion in response to external heat.

It is the prevailing rate of internal production of heat in man that largely determines his sensitivity to acute heat when he is faced suddenly with severe external warmth.

One can as yet only guess at the many bearings of variations in the vitamins that man requires, but thus far it is safe to say that where the climate is warm the vitamin B group is necessary in large quantities in order to maintain the optimal concentration for proper oxidative processes in tissues.

The great problem is that of how to deal with cyclonic storminess, for it is this that leads largely to the development of infections of the respiratory tract, especially in the north temperate zone.

Acute infections of the respiratory tract and acute rheumatic fever are predominantly diseases of stormy regions, being worst in the middle temperate belt of cyclonic storms and least troublesome in the calm regions of tropical warmth.

GORDON, Philadelphia.

SIMPLE METHODS FOR PERFORMING ARTIFICIAL RESPIRATION. RALPH M. WATERS, J. A. M. A. **123**:559 (Oct. 30) 1943.

Waters fully recognizes the value of the various manual maneuvers (the Schafer, the Silvester and their modifications) to accomplish artificial respiration but offers in this paper a brief review of some of the simple methods that can be applied by the use of ordinary devices.

In five figures he shows the equipment necessary and describes exactly how to fill the bag with oxygen and properly manipulate it so that the lungs will be filled and accurately emptied.

He comments on the intermittent inflation of the lungs with the subject face down, the head low and a pad under the "stomach"; with the handkerchief pulling the tongue so that an artificial airway may be gently passed, and finally with the subject lying face-up while the operator, holding the subject's nose closed, blows into the mouth intermittently, a handkerchief or other light material having been placed over the mouth to prevent contamination. The operator's right hand rests lightly on the chest in order that he may appreciate when the air is entering the lungs.

GORDON, Philadelphia.

"COLD VACCINES" AND THE INCIDENCE OF THE COMMON COLD. L. C. MCGEE, J. E. ANDES, C. A. PLUME and S. H. HINTON, J. A. M. A. **124**:555 (Feb. 26) 1944.

The authors briefly discuss the variables that occur in any given statistical study, with especial reference to the failure in several investigations to find a vaccine effective against epidemic coryza and related respiratory infections, as well as the confusion that is apt to arise when one attempts an exact interpretation of the results obtained.

For the purpose of obtaining accurate data the authors conducted their experiments on industrial workers, and they compiled the results from the records kept by the medical department of representative plants and offices participating in an attempt (*a*) to lower the amount of time lost because of respiratory infections by the use of vaccines and (*b*) to demonstrate the effectiveness of cold vaccines in an unselected industrial population if such effectiveness exists.

They summarize and conclude as follows: 1. Three vaccines for oral and two for parenteral administration currently offered as prophylactic against the common cold were given clinical trials in a group of industrial and office workers during the October to April seasons of 1941-1942 and 1942-1943. 2. No clearly evident protection against colds and related acute respiratory infections can be demonstrated in the results of this attempt at mass immunization. 3. The indiscriminate use of the cold vaccines now available is not the answer to the problem of industrial absenteeism due to acute respiratory infections.

GORDON, Philadelphia.

RESUSCITATION OF THE DROWNED TODAY. FRANK C. EVE, J. A. M. A. **124**:964 (April 1) 1944.

Eve discusses the merits of the Silvester, the Schafer and the Schafer-Nielsen methods of resuscitation of drowned persons. He comments on the value of the breathing machines.

In the body of his own presentation he makes this brilliant statement: "I submit that resuscitation is actually a trinity of ventilation, circulation and warmth directed to supplying warm blood, oxygenated by moving lungs, to the microscopic nerve cells which maintain respiration and tone." He describes in detail his rocking method: The patient is placed on a trestle and the legs are secured to the boards with bandages. The rocking is accomplished at the rate of ten double rocks a minute, with a tilt of 50 degrees to ventilate 600 cc. per rock (normal 500 cc.). The method was worked out in the Leeds physiology laboratory and later was adopted by the Royal Navy. He discusses in an interesting manner ventilation, circulation and warmth and concludes: "Resuscitation of the drowned is not merely working the bellows of the lungs but a fight to revive cold asphyxiated nerve cells by a circulation of warm blood oxygenated by moving lungs. Our old comfortable confidence in Schafer's method has been roughly shaken: Silvester's method is in many ways better, and the recent rocking method seems more promising still. Uncomplacently we must all 'go to school' again. . . . More experiments are badly needed: resuscitation is in the melting pot."

GORDON, Philadelphia.

VITAMINS AND THE EYE, EAR, NOSE AND THROAT: A REVIEW OF RECENT LITERATURE. ISAAC H. JONES, HAROLD S. MUCKLESTON, EUGENE R. LEWIS, WALTER P. COVELL and LELAND G. HUNNICUTT, Laryngoscope **53**:767 (Dec.) 1943.

Some astonishing cures have been attributed to vitamins in the past. The recent literature concerns mostly scientific work and few clinical results of the therapeutic use of the vitamins, particularly in regard to diseases of the eyes, the ears, the nose and the throat.

One recent advance is that concerning vitamin A and color blindness. At first the problem seemed a simple one, but later complexities occurred. Some results were confusing and contradictory, and other work indicated that vitamins B_2, D and C were also related to this condition.

Under the heading "Vitamins in General" the authors discuss the vitamin contents of foods and note that there is great variance in the vitamin content of a given food, depending on its preparation, cooking, source and other factors. The authors mention the valuable contributions of Price and Rosenberg to the subject of the vitamins. They discuss the relationship of vitamins K and C to hemorrhage. The relationship of vitamins to infection of the upper respiratory tract was studied by Cowan, Diehl and Baker. They concluded that in persons with an adequate diet the addition of any of the vitamins had no effect on the incidence or the severity of infections of the upper respiratory tract. The opposite is also true; that is, the lack of almost any of the vitamins, or of the other food elements essential for nutrition, will result in lowered resistance to infection. Likewise, vitamin A is closely associated with the health of the mucous membranes. Deficiency in this must be guarded against. Infection itself increases the need for vitamins. An adequate supply should be maintained during all infections.

The authors discuss the various vitamins in detail. In regard to vitamin B_1 they note that the upper limit of intake for safety is seldom reached in American diet. The development and the health of the teeth are influenced by the B complex, vitamin D and vitamin K. Palmer found that vitamin B_1 was very effective in migraine. Others found that moderate vitamin C deficiency was often encountered in medical practice. Pelner noted that there was a reduction in the sensitivity to salicylates and the sulfonamide compounds when vitamin C was administered in large doses. The tensile strength of wounds is higher in those having a high C content.

The authors discuss the intake of foods, pointing out that vitamins are merely activators. Without the proper foods or raw materials the activators are useless. The importance of an adequate intake of minerals is pointed out. Intake of the proper raw materials and vitamins is particularly important in pregnant women and infants.

HITSCHLER, Philadelphia.

VITAMIN AID IN THE TREATMENT OF COLDS—A PRELIMINARY REPORT. C. WARD CRAMPTON, New York State J. Med. **44**:162 (Jan. 15) 1944.

Crampton has been working with cod liver oil concentrate, administering 150,000 unit doses of vitamin A and 15,000 unit doses of vitamin D initially in twenty-four hours and a decreased dose thereafter; 103 persons used this concentrate "for more than a year"; 32, for more than two years. The large dose produces a "shock" resulting in rapid improvement of local and general symptoms. "In 24 hours sneezing, congestion, discomfort, pain, and discharge abate or disappear." Recurrence is possible at the end of the twenty-four hour period if the patient does not "lay up" or "if he exposes himself." Then 50,000 units of vitamin A is given but with less hope of immediate help. The method is merely an aid to other measures which are (1) to kill the infecting agent, (2) to increase the resistance of mucous membranes, (3) to use anti-infection agents (vaccines) and (4) to manage the physiologic processes of the body, such as digestion and nervous and circulatory control, efficiently. "These four factors are the four legs of a chair." "The remedy appears to do no harm. It is recommended for general clinical trial and direct and indirect scientific research." The author warns against assuming that this is a curative procedure, since no one remedy can hope to produce a "cure" but must supplement other agencies. An analytic table shows that, of 112 patients, 30.3 per cent were "cured," 51.8 per cent were "almost cured," 9.9 per cent were "better," 5.3 per cent showed "no change and 2.7 per cent were "worse"

VOORHEES, New York.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND PHILADELPHIA LARYNGOLOGICAL SOCIETY

GABRIEL TUCKER, M.D., *Chairman*

LOUIS J. BURNS, M.D., *President*

WILLIAM HEWSON, M.D., *Clerk*

THOMAS F. FURLONG, JR., M.D., *Secretary*

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Regular Meeting, Jan. 19, 1944

Suture of the Tonsillar Pillars as a Prevention of Hemorrhage in Local and General Tonsillectomies. Presented by DR. ISAAC B. HIGH, Reading, Pa. (by invitation).

The writing of this paper was prompted by the simplicity of the method and the remarkable results obtained. Only one laryngologist, Luc, of Paris, France, is an advocate of the method as I have used it. Luc advocates the suturing of the tonsillar pillars in adults, as well as in children, immediately after the removal of the tonsils; the suturing is easily performed and gives the patient and the operator security and tranquility of mind.

The suturing of the faucial pillars is simply a form of prolonged compression with a recession of the blood vessels into the tissues. It has been looked on and described as an operation of considerable difficulty because of the large denuded area and the inaccessibility as regards the placing of instruments and sutures in the tonsillar fossae.

Suturing of the faucial pillars, far from being a last resort, is a much easier method than finding the actual bleeding points and ligating them or suturing the pillars over a piece of gauze when hemorrhage occurs hours or days after operation. Temporary suturing of the pillars causes little discomfort at the time of operation and practically none after or during convalescence. Adults complain of no more pain than is felt when the pillars are gaping and the tonsillar beds exposed, and I have seen no more liability to adhesions or contractions than in cases in which suture was not performed.

This article is primarily a description of the suturing together of the faucial tonsillar pillars to prevent hemorrhage before it occurs. I have not endeavored to discuss the blood supply of the tonsils as a cause of hemorrhage, the technic of the surgeon as a factor in causing bleeding, the type of instrument (whether snare or guillotine), the many blood dyscrasias or hemorrhage (whether primary or secondary).

There is a saying among otolaryngologists that "if one does not perform tonsillectomies in great number, one does not have hemorrhage." I believe the performing of this number of operations on tonsils with immediate suturing together of the pillars and no history of hemorrhage is sufficient to merit a discussion of this simple and efficient procedure, which I most humbly invite.

DISCUSSION

DR. GEORGE M. COATES: I agree with Dr. High in everything that he has just said. It was many years ago that I first began to suture the pillars of the tonsils, not only to check obstinate oozing at the time of operation but also to provide against hemorrhage at a time when we at the Pennsylvania Hospital had to perform a great many tonsillectomies each afternoon that we operated. I could never see any objection to this method and, as Dr. High says, it is relatively easy, although when I first began to talk about it, it was condemned rather whole-heartedly by the older men in the profession.

There is less postoperative pain when this procedure is carried out since there are no raw surfaces to be irritated by food, the healing is more rapid than if the pillars remain widely separated, and the likelihood of infection is much reduced.

The question is always raised as to what happens to the fossae after removal of the tonsils and suture of the pillars. In my experience, the catgut dissolves in twenty-four hours or less and does not need instrumental removal. After that, the pillars usually separate quickly, and convalescence proceeds in the usual way.

Lautenschläger advocated this method in 1937 (Kirschner, M.; Lautenschläger, A., and Kleinschmidt, O.: Operative Surgery, Philadelphia, J. B. Lippincott Company, 1937, vol. 3). He stated that he passes the sutures through the anterior pillar, picks up the bed of the fossa with a pair of forceps, passes the suture behind this and brings it out through the posterior pillar. In all he uses three sutures, thus obliterating the tonsillar fossae. He uses silk instead of catgut and leaves the sutures in for from two to four days, but this has never seemed necessary to me since in most of our cases bleeding comes on within six to ten hours after the tonsillectomy. It is always essential to pass the sutures through the muscular structure of the pillars rather deeply in order to avoid cutting even in the few hours that they remain in situ, which will happen if just the edge of the pillar is picked up.

Reconstruction of the Frontal Bone with a Tantalum Implant, Following Removal of an Osteoma. Presented by CAPTAIN JOHN J. CONLEY, Medical Corps, Army of the Philippines (by invitation).

In February 1943 D. M., aged 22, white, on active military duty in North Africa, noticed a mild bulging of the right eye. This was associated with mild, intermittent frontal headaches and what he described as a failing of vision in his right eye. At this time he contracted an infection of the upper respiratory tract, and the right upper and lower eyelids were markedly edematous. The margin of the right frontal sinus was tender. This complication subsided promptly with conservative local intranasal treatment. At subsequent examinations the eye was still proptosed but the condition was gradually improving. There was no history of trauma or of chronic sinus disease.

Examination at this hospital showed the vision in both eyes to be 20/20. The fundi were normal; the

pupils were circular and equal and reacted to light and in accommodation. Although muscle balance was found to be within normal limits, the patient complained of intermittent diplopia. There were three old scars on the right cornea. The tension in both eyes was normal, and the proptosis in the left eye was 16 mm. and that in the right eye 23 mm. The visual fields were normal. The right upper lid was edematous and contained several superficial dilated tortuous veins. Further examination revealed a moderate prominence of the margin of the anterior plate of the right frontal sinus and marked bulging of the floor into the orbit. These areas were slightly tender to firm pressure. They were not red and did not appear inflamed. The airway in the nasal cavity was adequate. The mucous membrane was healthy, and there was no free pus. The middle meatus on the right side presented nothing unusual.

Roentgenograms revealed clouding of the right maxillary sinus and an osteoma in the right frontal sinus. The osteoma presented its most dense composition along its superior and lateral margins. The medial and inferior margins showed diploic structure. On July 26, with the patient under intratracheal anesthesia, the osteoma was removed.

On September 14 a large right frontal flap was made, the incision being kept within the hair line. The dura was hypertrophied, completely intact and of good color. The periosteum was adherent to it, and separation was tedious. As the brow on the right side was approached, the scar in its innermost dimension was adherent to the dura. Bleeding was controlled with a Bovie unit, and the previously prepared tantalum plate was placed in position. The edges of the bone about it were lowered in order to secure smooth approximation and to facilitate fitting. Three holes were made in the bone, one at the nasal margin, one at the extreme lateral margin and one superiorly. The thickness of the plate was $\frac{1}{3.000}$ inch (0.003 mm.), and it was fixed in position with tantalum wire. The plate did not rest against the globe or against the dura. However, there was a considerable dead space between the plate and the dura. The region of the nasofrontal duct was examined carefully, and the opening of the duct could not be entered with a probe. In addition, no blood escaped into the nose. The flap was reapproximated without drainage, and a moderately snug pressure bandage applied. The wound healed by primary intention, with little reaction, and the convalescence was uneventful.

DISCUSSION

MAJOR MARSHALL B. STEWART: Tantalum is a well known metal, not a mysterious substance. It has been used for a great many years on electrical needles for wood-burning purposes in the field of art. A great many substances have been used for restoring the contour of the cranial vault. Among them are the precious metals, celluloid, bone ivory, cartilage and vitallium (an alloy which has been introduced by the dental profession).

Dr. Lyndon Peer recently introduced "diced cartilage," which promises good cosmetic results. The neurosurgeons are responsible for the introduction of tantalum into this field. It has the advantage that it is the most inert of all the artificial substances. It does have some disadvantage, in that it is capable of transmitting thermal changes, as well as currents of diathermy during treatments.

Inasmuch as many substances have been introduced in the past twenty-five years for restoration, plastic materials such as the acrylic resins are being tried in this field. While tantalum is superior to most of the present artificial materials, its occasional use during the past few years may not warrant too early an evaluation of its universal use.

DR. HENRY A. MILLER: I have had no experience with the use of tantalum. I have used only tantalum wire. The number of cases in which I have used this, however, is not large enough to enable me to state whether it is superior to other preparations which have been used in maxillofacial surgery.

Typical Rhinoplastic Operation. Presented by DR. ROMEO A. LUONGO.

Preliminaries.—One hour before operation the patient is given 2 capsules—3 grains (0.19 Gm.)—of pentobarbital sodium. The vibrissae are clipped with manicure scissors, care being taken not to puncture the vestibular skin. The vestibules are scrubbed with medicinal soft soap U. S. P. and water on cotton applicators, four applicators being used on each side. The vestibules are then swabbed with alcohol followed by ether.

The nasal fossae are anesthetized: Dip 3 cotton-tipped applicators in a preparation of equal parts of a 10 per cent solution of a cocaine salt and a 1:1,000 solution of epinephrine hydrochloride, squeeze out excess fluid and insert as follows: one beneath the nasal dorsum along the olfactory sulcus (internal nasal nerve block); one at the foot of the middle concha (sphenopalatine ganglion block); one at the floor of the nose (nasopalatine and anterior palatine nerve block).

The patient is now placed on his back with the head elevated on a thin pillow. One or two drops of sterile castor oil are instilled into each eye.

Instructions for Personnel.—Second Assistant: Scrub hands. Don gloves but no gown. Drape the head and the neck of the patient with sterile linen. Withdraw the nasal applicators. Scrub the entire face including the vestibules with medicinal soft soap U. S. P. and water on gauze sponges for three minutes. Swab the face and the vestibules with alcohol followed by ether. Remove the gloves and don sterile raiment. Stand by.

First Assistant: Redrape the patient from head to foot. Arrange the instruments on a Mayo table. Take a position at the patient's left.

Operating Surgeon: Take a position at the patient's right. Pack the nasal fossae with long strips of half-inch gauze, leaving the vestibules clear.

Anesthetization of the External Nasal Pyramid.—Elevate the left ala with a rake retractor. Locate the plica nasi. Place a 2 inch (5 cm.) 24 gage needle at the center of the upper surface of this ledge. Pinch up the overlying skin with the left index finger and thumb. Under the control of these fingers advance the needle in the plane above the perichondrium and the periosteum to the nasal root. Discharge 0.5 cc. of the anesthetic solution—a 2 per cent solution of procaine hydrochloride to which has been added 5 drops to the ounce of a 1:1,000 solution of epinephrine hydrochloride (infratrochlear nerve block). Withdraw the needle to the junction of the nasal bone and the upper lateral cartilage and deposit another 0.5 cc. (external nasal nerve block). Repeat this procedure on the opposite side. Change to a 1½ inch (3.7 cm.) 24 gage needle. Introduce it intranasally at the outermost margin of the piriform opening. Carry to the point of an imaginary intersection of a vertical line drawn 1 cm. lateral to the inner canthus and an oblique line drawn from the

columella to the lateral canthus. Discharge 0.5 cc. of the solution as the needle is withdrawn (infraorbital nerve block). Repeat on the opposite side. Change to a 1 inch (2.5 cm.) 24 gage needle. Introduce this at the anterior nasal spine. Advance along the floor of the nose to the incisive foramen. On withdrawal introduce 0.5 cc. of the anesthetic solution (nasopalatine and anterior palatine nerve block). Repeat on the opposite side.

Uncovering of the Nasal Framework.—Elevate the left ala, using a rake retractor. Locate the plica nasi. With a Bard Parker no. 11 blade incise the aponeurosis connecting the upper and the lower lateral cartilage. Begin the incision at the center of the plica and extend it ventrally to the septum. Make a similar incision on the opposite side. Into the left incision introduce a straight double-edged pointed Joseph knife. Carry to a point 1 to 2 mm. above the caudal margin of the nasal bone in a plane immediately above the perichondrium of the upper lateral cartilage. Sweep the knife from side to side, separating the structures for a distance equal to the width of the blade, and withdraw. Repeat the procedure through the right intercartilaginous incision. Through the left incision introduce Fomon's curved double-edged blunt-tipped knife and continue the separation over the dorsum in the same plane as before. Repeat on the opposite side. Through the left incision introduce a MacKenty elevator and incise the periosteum 1 to 2 mm. above the caudal rim of the nasal bone. Substitute Joseph's periosteal elevator and gradually tease the periosteum from the dorsum up to the nasofrontal suture. Do not tear the membrane. Repeat on the opposite side. The soft tissues overlying the ventral and lateral nasal walls are thus freed. Introduce a button-end knife through the left incision and advance to the nasofrontal suture with the knife hugging the perichondrium of the upper lateral cartilage and passing beneath the periosteum of the nasal bone. Sweep obliquely downward across the dorsum, until the tip appears through the intercartilaginous incision in the right vestibule. Without altering the plane of the knife (which now transfixes the nose) cut to the caudal end of the septum. Turn the knife at a right angle, and with the blade hugging the caudal margin of the septal cartilage, divide the membranous septum through two thirds of its extent. Take a position at the head of the patient. With the straight Mayo scissors, held perpendicular to the nasal spine, straddle the membranous septum, the points of the scissors being made to lie on the nasal spine. With one snip of the scissors complete the separation.

Removal of the Hump.—Retract the left ala. Locate the incision in the plica nasi. Introduce the left bayonet saw. With the teeth directed dorsally, advance the instrument beneath the periosteum to the nasofrontal suture. Turn the teeth of the saw at a right angle. Check the position of the saw with the analyzed photographs and the cast, holding these in the same position as the patient's head. With the fingers of the left hand fix the apex of the saw. With the handle of the saw held in the right hand, the wrist and the elbow rigid, saw through the nasal bone, using the shoulder joint as a fulcrum. When the bone has been cut through, indicated by a loss of resistance, shift the handle obliquely and continue sawing through the perpendicular plate of the ethmoid sinus. When this has been accomplished, the tip of the saw will be felt at the nasal root on the opposite side. Remove the left saw. Retract the right ala and introduce the right saw the same as on the left. Fix the point of the saw in the niche at the root made by the left saw. Place the instrument against the right nasal bone at the same level as the saw cut on the left side. Check the level by palpating the left groove. Saw through the bone. Make sure that the bony hump has been cut through completely by moving it from side to side with the fingers. Introduce the MacKenty elevator and explore the separation. It will be found that the hump is free above and fixed below. Retract the left nostril. Introduce the short button-end knife into the vestibular incision. Insert the blade beneath the upper limit of the severed bony hump. Draw the knife down, cutting through the upper lateral and the septal cartilage, maintaining the same level throughout, until it falls free into the transfixion incision. Introduce the hump forceps through the left vestibular incision. Open the blades and grasp the hump so that one blade lies on either side of the segment. Extract. Instruct the assistant to exert pressure on the nose with a gauze sponge. Examine the hump. Make certain that the sides of the segment are symmetric. Any asymmetry in the sides of the bony hump indicate an error in technic. In such a contingency pass the Fomon rasp through the left vestibular incision. Place it obliquely across the dorsum and rasp from side to side until the plane of the dorsum is even. If the sides of the cartilaginous hump are asymmetric, introduce into the left vestibular incision a right-angled cartilage knife and pare the projecting side until it equals its fellow. Pass a sweeper through the left intercartilaginous incision to the root of the nose and gently remove the bone dust, spicules and other debris. Finally, introduce an angulated nasal speculum. Throw light into the nose and inspect the dorsum.

Shortening of the Medial Wall.—Insert a two-pronged retractor into the left nostril and draw the soft tissues to one side, exposing the septum and the upper lateral cartilages.

Inspect the septum and note the flare of the upper lateral cartilages.

Remove the retractor and introduce an angulated speculum between the soft tissues and the cartilaginous vault. Use this instrument as a guard.

With a Bard Parker no. 11 knife incise the remaining structures between the septum and the upper lateral cartilage, the blade cutting up on the angulated speculum.

Repeat on the opposite side. On inspection there will be seen three flaps, the septum in the middle and the upper lateral cartilages on either side.

Introduce a retractor into the left nostril and draw the soft tissues to one side to expose the septal cartilage.

Take a position behind the patient. Grasp the caudal end of the septal cartilage with fixation forceps. With straight Mayo scissors held perpendicularly and straddling the septum, excise a triangle with its base on the dorsum and its apex at the anterior nasal spine. The size of the base will govern the amount of shortening, as previously determined by analysis.

Approximation of the Upper Lateral Cartilages.—The upper lateral cartilages, which have been separated from the septum, are now reunited.

Retract the left ala and pass a half-curved-eye needle armed with no. 6-0 chromic gut through the upper (ventral) margin of the left upper lateral cartilage, from without in.

Carry the needle across the dorsum to the opposite site. Retract the right ala and pass the needle through the upper (ventral) margin of the right upper lateral cartilage, from within out.

Bring the gut ends out of the right nostril. Tie the first loop of a knot and exert slow, steady traction until the cartilaginous vault is of proper narrowness.

Make a second tie and cut the ends close to the knot.

Reenforce with a second suture with no. 6-0 chromic gut passed in like manner.

Shortening of the Lateral Walls (Upper Lateral Cartilages).—Before shortening the lateral walls (upper lateral cartilages) it is necessary to reapproximate the columella—freed from the septum by transfixion—to the septum. All other incisions are left open for purposes of drainage.

Pass a straight cutting needle armed with heavy silk (no. 1 or no. 2) through the full thickness of the ventrocaudal (anteroinferior) angle of the septal cartilage (perichondrium, cartilage, perichondrium). Continue the suture by passing the needle through the full thickness of the columella (vestibular skin, mesial crura, vestibular skin at junction of anterior and middle thirds).

Tie the first loop of a knot and exert tension on the ends of the thread. Note the gradual forward projection of the lobule.

When proper projection has been attained as determined by preoperative analysis, complete the tie.

Using the same needle and silk thread, reenforce the orthopedic stitch by passing another suture through the full thickness of the posterior third of the septum and the corresponding part of the columella.

Retract the ala with a rake retractor and note the projection of the upper lateral cartilage into the vestibule.

Grasp the protruding portion with a rat tooth forceps but exert no traction. With angulated scissors, excise the projecting part flush with the incision.

Repeat on the opposite side.

Narrowing of the Nose.—Flatness of the dorsum following removal of a hump is eliminated by shifting the lateral walls to the midline. This is accomplished by (1) removal of solid bony wedges, as the root, and (2) lateral osteotomies through the frontal processes of the maxillas.

Removal of a Bony Wedge.—Engage a Fomon guarded chisel at the junction of a solid block of bone and the frontal process of the maxilla. With a few taps of a mallet on the chisel held obliquely, cut through the bone to the nasofrontal articulation.

Engage the chisel at the junction of a solid block of bone and the perpendicular plate of the ethmoid sinus. Cut through obliquely so that the cuts made with the chisel meet beneath the wedge.

Extract the bony wedge with a stout hemostat.

Repeat on the opposite side.

Lateral Osteotomy.—Retract the left ala. Locate the lateral margin of the apertura piriformis (point below the alar-facial junction). With a Bard Parker no. 11 knife incise the overlying mucosa down to the bone.

Introduce a Joseph elevator beneath the periosteum and raise the overlying tissues en masse to a point midway between the nasion and the inner canthus. The extent of this elevation should be no more than will admit a saw comfortably.

Introduce a grooved director into the incision made along the margin of the piriform opening. Using this as a guide, pass left a right-angled saw into the periosteal pocket, the teeth directed downward. Remove the grooved director. Turn the saw so that the teeth lie at a right angle to the bone. Manipulate the instrument until it falls into the nasofacial groove, the apex lying at a point midway between the nasion and the inner canthus and the base at the outermost margin of the piriform process. Place the fingers of the left hand over the apex of the saw to fix it and protect the inner canthus.

With two or three up and down strokes make a groove in the bone. Elevate the handle and cut through the upper part of the frontal process. Gradually lower the handle and cut through the caudal margin of the frontal process.

Deepen the groove through about one-half thickness of the bone. Under no circumstances cut all the way through.

Repeat the process on the right side in exactly the same manner.

Through the left intercartilaginous incision pass the Fomon guarded chisel until it abuts the apex of the space left after the removal of the bony wedge. With a few taps of the mallet engage the chisel at the nasofrontal articulation. Brace the instrument against the lateral nasal wall, exert lateral pressure and fracture the bone outward.

Repeat on the opposite side.

Take a position on the left side of the patient. Place a pad of gauze over the left nasal wall. Brace both thumbs over the gauze, spread the fingers over the opposite zygoma and with moderate pressure force the outfractured nasal walls to the midline. Repeat on the opposite side. A complete fracture will be evidenced by a short, decisive snap. If a greenstick fracture occurs, the sound will be less distinct and the bones less movable, with a tendency to spring back to place.

Modeling of the Lower Lateral Cartilages.—Notice that the lobule, even if in proper relationship with the balance of the nose before operation, is now relatively too wide and too projecting and must be modeled to harmonize with the reconstructed osseous and upper cartilaginous vault.

Retract the left ala and with a Bard Parker no. 15 knife make a circumferential incision through the vestibular skin along the lower margin of the lower lateral cartilage.

Introduce blunt double-edged scissors between the lower lateral cartilage and the overlying structures, staying on the plane immediately above the perichondrium.

Gradually separate the tissues in an upward (cephalad) direction until the incision previously made in the plica nasi is reached, indicated by loss of resistance.

Carry the scissors obliquely and gradually separate the overlying structures until the angle of the lower lateral cartilage is completely freed.

Repeat the procedure on the opposite side so that separation over the entire lobule is complete.

Insert a dural hook beneath the angle of the left lower lateral cartilage and draw the cartilage out of the nostril. Under this double-pedicled vestibular skin-cartilage flap pass a strabismus hook.

Expose the right lower lateral cartilage in like manner. With cartilages thus exposed, further manipulation will depend on the character of the deformity. Assuming that there is a projecting bulbous tip, proceed as follows:

Cut through the cartilage on a line medial to the angle.

With a dural hook steady the left lateral flap (lateral crus) and with fine curved scissors remove a

section from the free margin, of such a size as to bring about the desired reduction in projection. Continue paring upward along the cephalic margin of the cartilage, tapering off toward the pedicle to reduce the bulbousness.

Draw out the mesial crus and round off the cut margins.

Repeat on the opposite side.

The two cartilaginous flaps on each side will fall together, and the reduction of width of the nasal tip will equal the combined width of the sections of cartilage excised.

Dressing.—The assistant applies pressure over the nose while a splint is being prepared.

Cut flannel in the form of a butterfly and cover it with softened stent.

Mold the flannel-lined stent to the nose.

Hold in place until it hardens.

Fix with strips of half-inch adhesive tape running obliquely from forehead to cheek and horizontally from cheek to cheek. Strap a small pad of gauze over the base of the nose to absorb any ooze.

Postoperative Management.—During the first forty-eight hours keep the patient flat on his back. Apply sand bags to each side of the head. Prescribe a semi-solid diet for the first twenty-four hours. Allow no visitors. Advise against talking, laughing and like movements. Have the hands restrained at night. For discomfort or restlessness give phenobarbital 1 to 2 grains (0.06 to 0.13 Gm.), codeine ¼ grain (0.016 Gm.) or acetylsalicylic acid 5 to 10 grains (0.32 to 0.65 Gm.). Visit the patient twice daily. Note the temperature and the pulse and respiratory rates at each visit. A rise of temperature of 1 degree with a gradual fall within the first forty-eight hours is of no consequence.

On the second day after operation remove the splint. With an applicator dipped in benzene gradually separate the adhesive. Carefully lift the splint from the nose, working from above downward.

Instil a few drops of sterile oil or hydrogen peroxide into each nostril and gently withdraw the bismuth tribromphenate packs. Guard against pulling as it may displace the flaps. If gauze sticks despite the use of oil or peroxide, leave it in for another day.

Cover the nose with liquid petrolatum. Cover with a pad of gauze and palpate. At this stage one can shift nasal elements and reassemble them should they be found out of alinement.

On the fifth or sixth day remove the two sutures.

DISCUSSION

DR. SAMUEL FOMON (by invitation): Dr. Romeo A. Luongo's excellent description of a typical rhinoplasty leaves little for a commentator to add in the way of technic. Seldom, however, is one confronted with a nose that requires a so-called typical rhinoplasty such as Dr. Luongo has just presented. I am sure that in his admirable step by step description of the procedure he attempts merely to illustrate the basic principles of the technic involved in rhinoplastic surgery. Owing to the endless variety of nasal deformities, each case is a problem unto itself.

All conversant with this type of surgical operation admit that the technic requires no high order of skill. Successful results depend not so much on manual dexterity as on proper evaluation of the causal factors. Let me illustrate with two common deformities:

The first is a recessed lobule. No single operative procedure will be effective in all cases. If a recession is simulated owing to an abnormal projection of the nasal dorsum, a reduction of the nasal pyramid is all that is necessary to eliminate the illusion of recession. Obviously, to build up the lobule to harmonize with the unduly projecting dorsum in such a case would serve only to exaggerate the deformity.

If a short columella is the cause, the only recourse is to lengthen the columella at the expense of the upper lip and thus allow the lower lateral cartilages to assume their normal positions.

If the recession is due to lack of septal support, correction can be accomplished only by the introduction of a cartilaginous strut into the columella.

The lower lateral cartilages may be dislocated backward, in which case they must be separated from the upper cartilaginous vault, brought forward and fixed to the septum in their normal positions.

Atrophy of the lower lateral cartilages may account for the deformity. Here forward extension is obtained in one or the other of two ways: by cutting the lateral crura, swinging them forward and fixing them in a more anterior position; or by implanting a button of cartilage subcutaneously over the angles of the lower lateral cartilages.

The second deformity that I would cite is the projecting lobule. Here again the condition may be simulated. If it is due to a recession of the dorsum, elevation of the dorsum to form a normal profile is all that is necessary.

If it is due to a projecting ventral margin of the septal cartilage, excision of the protruding portion will permit the lower lateral cartilages to position themselves properly.

When it is caused by abnormally long mesial and lateral crura, these structures must be reduced.

When one considers that these are but a few of the causal factors and that they not infrequently occur in combination, it can be readily appreciated that the rhinoplastic operation is not so much one of technic as one of diagnosis.

DR. JOHN D. REESE: I enjoyed Dr. Luongo's splendid presentation of the details encountered in the reconstruction of a typical nasal deformity. The pictures he presented and the diagrammatic drawings in color were well executed; they present clearly both the anatomy involved and the salient details of the indicated surgical procedure.

I am a little surprised by the complete candor with which Dr. Luongo represents the mobilization and sectioning of the lateral cartilages of the nose—both upper and lower. Cartilage is a temperamental structure as regards form, which can be altered only by complete section and then there is no union of the disjoined portions. Both the upper and the lower lateral nasal cartilages function as a sort of cantilever canopy for the alae—preventing collapse of the alae on inspiration. I speak a word of caution as to drastic section of these two structures, especially of the lower cartilages, as difficulties may be encountered in the function of the alae, and I know of no more difficult condition to correct satisfactorily in a cosmetically conscious patient.

DR. SAMUEL COHEN: The animated drawings shown by Dr. Luongo are interesting. The order in which they are put might be altered and I think for the better. The use of the chisel on each side of the nasal septum in order to make the fracturing at the naso-glabellar angle more accurate has been practiced by many rhinologic surgeons for some time now. If in addition to this a thin sliver of bone is punched out in this region when the bony bridge is wide, a better

narrowing of the bridge results. Let me stress that narrowing the bony part of the nose between the eyes and deepening the nasoglabellar angle are procedures that still need improvement.

The method of inducing anesthesia that I use differs from that demonstrated. I believe that it is an improvement in that the nasal contour is not obscured by swelling due to the injection of procaine hydrochloride. The advantages of not obscuring the area to be worked on are so obvious that I need not dwell on them. I have used this technic for several years, and this is the first time I have discussed it in public.

Four cottoned applicators dipped in a 5 per cent solution of a cocaine salt are inserted intranasally, one in each nostril between the middle turbinate and the septum and one behind the posterior end of each middle turbinate. The applicators are left in for five minutes. One is now ready to inject the solution of procaine hydrochloride. A 5 cc. syringe with a 2 inch (5 cm.) needle, gage 13, and a 2 per cent solution of procaine hydrochloride with 10 drops of epinephrine to the ounce (29 cc.) is used.

I make only two needle punctures of the skin, one in each cheek about ¼ inch (6 mm.) lateral to each alar groove. The needle is inserted into the cheek, and a few drops of solution are injected. The needle is then directed to the infraorbital foramen and made to enter the infraorbital nerve. The needle is now drawn back until about one quarter of it is left in the cheek; it is then directed upward along the side of the nose until it reaches slightly anterior to the inner canthus. As the needle is directed upward one tries to hug the bone as closely as possible and inject the solution slowly. The needle is again withdrawn until about one quarter of it is left in the cheek, and now it is turned toward the upper lip and pushed in that direction until it just passes the columella-lip junction. The solution is injected as the needle goes toward its goal. About 2 or 3 cc. of the solution of procaine hydrochloride is used. The procedure is now repeated in the opposite cheek.

When the injection of the anesthetic solution is finished, a slight swelling of tissue is noted from each inner canthus to each alar groove. Anesthesia is now complete. There is no necessity to infiltrate the region of the tip, so one does not obscure the lower lateral cartilages. This is important as the tip needs careful surgical attention if one is to obtain satisfactory end results in this difficult area. The absence of swelling has been, at least to me, of great help in dissecting the lower lateral cartilages.

I was glad to hear Dr. Fomon stress the need of careful diagnosis of the individual abnormalities in each case and of careful preparation of an operative blueprint before operating. Too much attention cannot be paid to this point. Its value cannot be overestimated.

The operation which Dr. Fomon described for the correction of a dislocated septal cartilage was first described by me many years ago and discussed in subsequent papers. In this procedure, in addition to separating the cartilage from its usual bed, scraping the vomer and removing a sliver of cartilage submucously above the bend, an important point was missed. The attachment of the upper lateral cartilages on each side of the septum must be cut; otherwise the dislocated angulated piece of septal cartilage cannot be properly swung into the correct position, and even if sutured, it would not stay in that position. It is only when the dislocated septal cartilage is made to swing freely and easily into the desired new position that it will stay when sutured. I have performed this operation on very young children with gratifying physiologic and cosmetic results.

It gives me great pleasure to see the interest that is now being shown by otolaryngologists in procedures for the correction of nasal contour. This interest promises to bring good results to all concerned. The field needs a great deal of research, and I am sure the interest now displayed will bring great improvements in our technic.

DR. THOMAS F. FURLONG: I enjoyed the papers by Dr. Luongo and Dr. Fomon. They received first prize for their exhibit at the meeting of the American Academy of Ophthalmology and Otolaryngology.

DR. MATTHEW S. ERSNER: I should like to compliment Dr. Luongo on the interesting as well as important subject which he presented this evening. The technic of a typical rhinoplasty as presented by Dr. Luongo is an outgrowth of the Fomon School of thought. Unfortunately, few cases can be catalogued as cases in which a typical rhinoplasty is to be done. When a careful analysis and diagnosis are made and the proper surgical correction is instituted, the cosmetic results are universally good. This is so because the technic is simple.

The qualified rhinologist should make every effort to learn rhinoplasty, because the performance of intranasal as well as extranasal surgical corrections is in his domain.

Book Reviews

Tuberculosis of the Ear, Nose and Throat, Including the Larynx, the Trachea and the Bronchi. By Mervin C. Myerson, M.D., New York. Price $5.50 postpaid. Springfield, Ill.: Charles C Thomas, Publisher, 1943.

Dr. Myerson presents his textbook on tuberculosis of the ear, the nose and the throat, including the larynx, the trachea and the bronchi. Much is compressed in 291 pages. The printing, the format and the binding, as well as the arrangement of the material, are all first class.

The author bases his treatise on his own observations gleaned from years of experience in this particular field. Controversial issues are fully discussed. In nearly every chapter he frankly and sincerely states his opinion on either the etiologic factors or the therapy of a given condition, and his arguments are so logical and rational that they merit the reflection of the otolaryngologic world. He makes a strong plea for therapy based on sound concepts of pathologic anatomy and physiology, and deplores haphazard measures.

Many of the older ideas are presented in a new light. Each chapter is followed by a bibliography which is valuable in that it helps the reader to search further a particular subject. There are 88 figures in all; some are photomicrographs, while others are charts, drawings, photographs and schemas. All of these supplement the text matter and clarify no little the already lucid presentation in the text. Interesting historical data are given in the beginning of each chapter.

This textbook is strongly recommended to all otolaryngologists, as well as to graduate students and resident physicians in otolaryngology. It is so well written that it may be extremely valuable as a consultation book on tuberculosis of the ear, the nose and the throat, including the larynx, the trachea and the bronchi, to specialists and students of tuberculosis, as well as to internists and pediatricians.

Minor Surgery. Edited by Humphry Rolleston and Alan Moncrieff. Price $5. New York: Philosophical Library, 1944.

This textbook totals 174 pages. It contains 18 chapters, each one devoted to a specialty. The chapter on the nose and the throat consists of 7 pages and gives a sketchy outline of nasal examination, induction of anesthesia, procedures in the treatment of fracture of the nasal bones, nasal hemorrhage, foreign bodies and tonsillar abscess and tonsillectomy. The chapter on the ear consists of 9 pages and covers the following: removal of wax, syringing for debris, removal of foreign bodies from the meatus, treatment of furunculosis, inflation of the middle ear (Valsalva's method, Politzer's method and catheterization of the eustachian tube) and myringotomy.

It is the opinion of this reviewer that this text is not to be recommended to otolaryngologists, for each reader of the ARCHIVES OF OTOLARYNGOLOGY will welcome only a text that is authoritative, comprehensive, informative and functional.

News and Comment

DIRECTORY OF MEDICAL SPECIALISTS

The third edition of the Directory of Medical Specialists, listing names and biographic data of all physicians certified by the fifteen American Boards, is to be published early in 1945. The certified specialists are requested to make prompt return of the notices regarding their biographies as soon as possible after receiving the proper forms, soon to be mailed from the publication office.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.

President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.

Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

Place: Palmer House, Chicago. Time: Oct. 8-11, 1944.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland, Ohio.

Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.

Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Oram R. Kline, 414 Cooper St., Camden, N. J.

Southern.—Chairman: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.

Middle.—Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.

Western.—Chairman: Dr. Aubrey G. Rawlins, 384 Post St., San Francisco.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.

Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

THE ARCHIVES OF OTOLARYNGOLOGY is published by the American Medical Association to stimulate research in the field of otology and laryngology and to disseminate knowledge in this department of medicine.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. George M. Coates, Chief Editor, 1721 Pine Street, Philadelphia, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF OTOLARYNGOLOGY. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones will be supplied by the Association when the original illustrations warrant reproduction and when their number is not considered excessive.

Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

Matter appearing in the ARCHIVES OF OTOLARYNGOLOGY is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the ARCHIVES OF OTOLARYNGOLOGY or in any of the other publications issued by the Association will not be permitted.

The ARCHIVES OF OTOLARYNGOLOGY is issued monthly. The annual subscription price (for two volumes) is as follows: domestic, $6.00; Canadian, $6.40; foreign, $7.00, including postage. Single copies are 75 cents, postpaid.

Checks, money orders and drafts should be made payable to the American Medical Association.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (three volumes): domestic, $8.00; Canadian, $9.50; foreign, $12.00. Single copies, 25 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): domestic, $5.00; Canadian, $5.40; foreign, $6.00. Single copies, 75 cents.

AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. Includes carefully prepared reviews, based on recent pediatric literature, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.50. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and abstracts of the literature on these two subjects, transactions of the important dermatologic societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.50. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents, except special numbers.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of pathology. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents, except special issues.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

WAR MEDICINE—Monthly. Devoted to material on preparedness and scientific advancement in relation to military, naval, public health and allied services. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: domestic, $12.00; Canadian, $14.00; foreign, $14.00.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **CHICAGO 10**

SEP 14 1944

CONTENTS

PAGE

Intracranial Suppuration Secondary to Disease of the Nasal Septum: A Survey of the Literature; A Report of Cases and Animal Experiments. Captain Lloyd K. Rosenvold, Medical Corps, Army of the United States 1

Chemotherapy and Biotherapy: Their Relation to the Prevention and Treatment of Diseases of the Ear, Nose and Throat. John A. Kolmer, M.D., Philadelphia 17

Modern View of Neuralgia Referable to Meckel's Ganglion: Report of Cases Showing Relief of Pain and Sometimes Arrest of Development of Ulcers of the Cornea by Cocainization of the Ganglion. B. R. Dysart, M.D., Pasadena, Calif. 29

Thrombosis of the Cavernous Sinus with Hemolytic Streptococcic Bacteremia: Treatment by Intravenous Injection of Sulfadiazine and Penicillin, with Recovery. Jonas W. Wolf, M.D., Yonkers, N. Y. 33

Present Status of Diagnosis and Management of Ménière's Syndrome. Hans Brunner, M.D., Chicago 38

Absorptive Capacity of the Nose. Maurice Saltzman, M.D., Philadelphia 44

Cancer of the Trachea: Report of Five Cases. Gilbert E. Fisher, M.D., Birmingham, Ala. 49

Prevention of Traumatic Deafness: Further Studies. Captain William H. Wilson, Medical Corps, Army of the United States ... 52

Clinical Notes; New Instruments and Technics:

Modification of the Blade of the LaForce Tonsillectome. Frederick E. Keller, M.D., Philadelphia 59

Case Reports:

An Unusual Foreign Body of the Larynx. Major Arnold F. Judge, Medical Corps, Army of the United States 60

Adamantinoma of the Maxillary Sinus. Walter F. Mosher, M.D., Ventura, Calif. 61

Abstracts from Current Literature 63

Society Transactions:

College of Physicians of Philadelphia, Section on Otolaryngology, and Philadelphia Laryngological Society 67

Book Reviews 73

News and Comment 73

Directory of Otolaryngologic Societies 74